UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF ILLINOIS
EASTERN DIVISION

IN RE: ABBOTT LABORATORIES, ET AL., PRETERM INFANT NUTRITION PRODUCTS LIABILITY LITIGATION	**MDL NO. 3026** **Master Docket No. 22 C 00071**
This Document Relates to: **All Cases**	**Hon. Rebecca R. Pallmeyer**

MEMORANDUM OPINION AND ORDER

Plaintiffs in this multi-district litigation (MDL) assert that Defendants Abbott Laboratories ("Abbott") and Mead Johnson Nutritional Company ("Mead Johnson") manufactured cow's-milk-based formula products ("CMBF")[1] that caused infants to develop necrotizing enterocolitis ("NEC"), a devastating disease affecting pre-term infants. As part of these consolidated proceedings, Defendants have retained experts to testify in bellwether cases chosen by the parties to proceed to trial before this court.[2] Plaintiffs now move [612] to exclude five of these experts—three disclosed by Abbott, two by Mead Johnson—under Federal Rule of Evidence 702 and *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579 (1993). These experts include Mead Johnson's epidemiological expert for the *Inman* bellwether, Dr. Larry Hedges; Mead Johnson's neonatology and pediatrics expert for the *Inman* bellwether, Dr. Erika Claud; Abbott's epidemiological expert for the *Mar* and *Etienne* bellwethers, Dr. Robert Makuch; Abbott's

[1] As noted in the court's prior order, terminology regarding the specific products at issue in this MDL varies, with some experts grouping all cow's-milk-based products (formula and fortifier) into one term, others referring only to formula products. (Order [646] at 1 n.1.) For consistency in this opinion, and because the infants in the bellwether cases were primarily fed formula (not fortifier), the court will use "CMBF" to describe the class of products at issue and will note the distinction between formula and fortifier where relevant.

[2] On October 4, 2024, the parties agreed by stipulation that the following cases would be bellwether cases proceeding to trial in this court: *Mar v. Abbott Lab'ys*, No. 22 C 232 (hereinafter "*Mar*"); *Diggs v. Abbott Lab'ys*, No. 22 C 5356 (hereinafter "*Diggs*"); *Etienne v. Abbott Lab'ys*, No. 22 C 2001 (hereinafter "*Etienne*"); *Inman v. Mead Johnson & Co., LLC*, 22 C 3737 (hereinafter "*Inman*").

epidemiological expert for the *Diggs* bellwether,[3] Dr. Brian Smith; and Abbott's health economics expert for all bellwethers, Dr. Amanda Starc. For the following reasons, Plaintiffs' motion is granted in part and denied in part.

BACKGROUND

For purposes of this opinion, the court assumes general familiarity with the facts underlying the claims in this MDL, as well as with the court's prior orders in this proceeding. In an earlier ruling [646] (hereinafter "General Causation Order"),[4] the court denied Defendants' joint motion to exclude Plaintiffs' general causation experts. That ruling provides background as to the nature of Plaintiffs' epidemiological and biological theories for how CMBF causes NEC. The court's order granting Abbott's motion for summary judgment in *Mar* ([96] in No. 22 C 232)[5] provides a representative fact pattern for the claims against Abbott and Mead Johnson.

LEGAL STANDARDS

Federal Rule of Evidence 702 governs the admissibility of expert testimony. It states:

> A witness who is qualified as an expert by knowledge, skill, experience, training, or education may testify in the form of an opinion or otherwise if:
>
> (a) the expert's scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue;
>
> (b) the testimony is based on sufficient facts or data;
>
> (c) the testimony is the product of reliable principles and methods; and
>
> (d) the expert has reliably applied the principles and methods to the facts of the case.

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[3] The court has now granted summary judgment in favor of Abbott in the *Diggs* bellwether but will nevertheless address Plaintiffs' challenges to Dr. Smith's testimony as his general causation testimony may have application to other cases in the MDL.

[4] *In re Abbott Lab'ys Preterm Infant Nutrition Prods. Liab. Litig.,* No. 22 C 00071, 2025 WL 1283927 (N.D. Ill. May 2, 2025)

[5] *In re Abbott Lab'ys Preterm Infant Nutrition Prods. Liab. Litig.*, No. 22 C 00071, 2025 WL 1282749 (N.D. Ill. May 2, 2025).

FED. R. EVID. 702. In *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579 (1993), the Supreme Court recognized that Rule 702 creates a "gatekeeping role for the judge" by "assign[ing] to the trial judge the task of ensuring that an expert's testimony both rests on a reliable foundation and is relevant to the task at hand." 509 U.S. at 597. Under *Daubert*, "the district court must evaluate: (1) the proffered expert's qualifications; (2) the reliability of the expert's methodology; and (3) the relevance of the expert's testimony." *Gopalratnam v. Hewlett-Packard Co.*, 877 F.3d 771, 779 (7th Cir. 2017). In determining whether an expert's methods are reliable, courts look to factors including, but not limited to, "(1) whether the scientific theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether a particular technique has a known potential rate of error; and (4) whether the theory or technique is generally accepted in the relevant scientific community." *Schultz v. Akzo Nobel Paints, LLC*, 721 F.3d 426, 431 (7th Cir. 2013) (citing *Daubert*, 509 U.S. at 593–94). The party seeking to introduce the expert witness testimony bears the burden of demonstrating its admissibility. *Gopalratnam*, 877 F.3d at 782.

DISCUSSION

Plaintiffs' omnibus motion raises various arguments against the different experts proffered by Abbott and Mead Johnson; the arguments do not challenge the experts' respective qualifications, but rather target specific methods used by the experts or the experts' application of these methods to the facts of the bellwether cases. In this opinion, the court addresses the specific arguments raised by Plaintiffs in their motion, with the understanding that *Daubert* rulings are provisional and subject to later review as the bellwether cases develop.

I. Dr. Larry Hedges

Dr. Hedges is a biostatistician hired by Mead Johnson to respond to Plaintiffs' epidemiology expert, Dr. Logan Spector, and perform a meta-analysis of epidemiological studies testing the relationship between CMBF and NEC. His testimony is offered as part of the *Inman* bellwether proceedings.

A. Dr. Hedges' Report

1. Qualifications

Dr. Hedges has served as the Board of Trustees Professor of Statistics at Northwestern University since 2005, where he also serves as Professor of Education and Social Policy, Psychology, and Medical Social Sciences. (Hedges Rep. [612-2] at 1.) He is also a faculty fellow in the Institute for Policy Research. (*Id.*) He has a B.A. in mathematics from the University of California, San Diego, an M.S. in statistics from Stanford University, and a Ph.D. in mathematical methods in educational research from Stanford in 1980. (*Id.*) Before joining the Northwestern faculty, between 1980 and 2005, he was a faculty member at the University of Chicago, rising from Assistant Professor to the rank of Distinguished Service Professor. (*Id.*)

Dr. Hedges has authored or co-authored more than 250 research papers and has co-authored a seminal book on using statistical methods in meta-analysis. (*Id.*) He has also edited three handbooks on systematic literature reviews and co-authored editions of a textbook on meta-analysis. (*Id.*)

2. Methodology

Dr. Hedges was engaged by Mead Johnson to "review the meta-analysis publications that exist, review a meta-analysis performed by Plaintiffs' expert Dr. Spector in this litigation, and conduct a meta-analysis of the research studies included in Dr. Spector's report" that examine an association between CMBF and NEC in pre-term infants. (*Id.* at 2.) The report Dr. Hedges prepared has three sections: (1) an analysis of existing reviews (publications discussing studies), (2) an analysis of the studies contained in Dr. Spector's report, and (3) a discussion of whether the available studies support a finding of causality between CMBF and NEC using the "Bradford Hill" factors (described more fully below). For the first two sections, Dr. Hedges performed his own set of meta-analyses: in analyzing existing reviews, Dr. Hedges conducted independent meta-analyses of the underlying studies discussed in the reviews; in reviewing Dr. Spector's

report, Dr. Hedges conducted independent meta-analyses of the studies selected for review by Dr. Spector. The court briefly describes Dr. Hedges' methods in performing meta-analyses before turning to his application of those methods to the existing reviews and Dr. Spector's report.

a. Dr. Hedges' Meta-Analysis Methods

In performing his meta-analyses in this case, Dr. Hedges used a method known as the "Knapp-Hartung method" or "Knapp-Hartung adjustment." (*Id*. at 9–10.) As Dr. Hedges explains, there are two fundamental "models" of meta-analysis methods: common-effect models and random-effect models. (*Id*. at 9.) Common-effect models assume that the underlying association is identical in the data set (e.g., all studies observe a positive association between exposure and disease, or all studies observe a negative association) and present a weighted average association across the studies. (*Id*.) As such, common-effect models largely ignore variations in the observed associations in the underlying data set.[6] (*Id*.) Such models are appropriate, Dr. Hedges explains, where there is reason to believe that results will be identical across all studies—for example, because each study follows an identical design. (*See id*.) Random-effects models, on the other hand, assume a level of variation in the association observed across the studies, and incorporate an estimation of variation into the meta-analysis. (*Id*.) Such models are more appropriate for a meta-analysis of studies where treatment or study designs differ across the various studies. (*Id*.) The Knapp-Hartung method is one kind of random-effect model that introduces an empirical calculation of variation between the studies, and "generally produces more accurate tests of statistical significance and confidence intervals than other methods" for small samples. (*Id*. at 10.)

[6] For example, if there are two studies observing the association between substance A and disease B, where one study observes a strong positive correlation between A and B and the second study observes a strong *negative* correlation between A and B, a common-effect model will produce a weighted average of the two associations (some number in between the two reported results), though the studies produced significantly different results.

In addition to using the Knapp-Hartung method to produce average risk ratio values across various studies, Dr. Hedges' meta-analyses also considered heterogeneity (that is, variations in the results reached in studies that are the subject of the meta-analysis). Some variation is to be expected, but great variation (that is, substantial heterogeneity) detracts from the strength of the observed association. Dr. Hedges' first method of calculating heterogeneity was the *Q*-test, which compares the variation in observed associations across studies to expected variations in estimation errors[7] (also known as "background noise"), and represents this comparison as a numerical "*Q*-statistic." (*Id.*)[8] In other words, the *Q*-test represents whether the variation in results in the underlying studies is consistent with expected variations in data sets (low *Q*-statistic, no inference of heterogeneity), or if the variation is incompatible with mere chance or "noise" (high *Q*-statistic, high inference of heterogeneity). (*See id.*); *see also* Cochrane Handbook for Systematic Reviews of Interventions, Section 10.10.2, https://training.cochrane.org/handbook/current/chapter-10#section-10-10-2 (last accessed Aug. 6, 2025). Dr. Hedges' second method of calculating heterogeneity was to measure the absolute magnitude of variation of "true effects,"[9] or the standard deviation of study results (accounting for estimation error) from the

[7] As Dr. Hedges explains, expected variations in estimation errors is a function of the sample size of the set of studies—groups of studies with small sample sizes will provide large variations in estimates, while large sample sizes will have smaller expected variations in estimates. (Hedges Rep. at 10–11.)

[8] In his deposition, Dr. Hedges explains that a *Q*-statistic's significance depends on whether the value is "bigger than whatever the critical value for the number of studies at hand." (Hedges Dep. Tr. at 97:21–98:2.)

[9] In calculating the heterogeneity of various studies, Dr. Hedges distinguishes between the "true effect" and the "estimate" reported by the study. (Hedges Rep. at 12.) The "estimate" refers to the association reported on the face of the study—it is considered an estimate in that any statistical analysis will likely involve some uncertainty. (*Id.*) The "true effect" reported by a study, on the other hand, is found by taking the estimate reported by the study and subtracting the "estimation error." (*Id.*) The estimation error for any given study cannot be determined in the abstract, but statistical theory has developed ways of measuring the variation in estimation errors as a function of sample size. (*Id.*) Thus, while Dr. Hedges cannot look at a group of studies and determine a "true effect" of each study, he can calculate the average variation in true effects by taking the variation of estimates (which can be observed on the face of the studies) and subtracting the variation in estimation errors (provided by statistical theory). (*Id.*)

average effect, denoted by the Greek symbol tau (τ). (Hedges Rep. at 11.) Dr. Hedges' third measure of heterogeneity looked at "relative heterogeneity"—the absolute variation of true effects (*see supra* n. 9) relative to the variation in estimates (the values reported by the studies). (*Id*.) Relative heterogeneity, represented as a percentage by the "I^2 statistic," represents the extent to which variations in observed effects are due to real differences in results rather than chance. (*Id*.); *see also* Cochrane Handbook, Section 10.10.2. https://training.cochrane.org/handbook/current/chapter-10#section-10-10-2 (last accessed Aug. 6, 2025). Finally, Dr. Hedges also calculated prediction intervals as a fourth measure of heterogeneity; prediction intervals use sampling uncertainty and the variation between studies to predict the range of results that a new study might produce. (*Id*. at 11–12.)

b. Analysis of Existing Reviews

Dr. Hedges' report discusses three systematic reviews discussing the effects of CMBF on NEC incidence in preterm infants: Premkumar 2019, Miller 2018, Quigley 2019, and Quigley 2024. (*See id*. at 12–14.) The report does not explain how Dr. Hedges chose these specific publications for review or whether they represent an exhaustive list of systematic reviews on the association between CMBF and NEC. In any event, for each review, Dr. Hedges briefly explains the findings of the review before performing a meta-analysis (where possible) of the random-controlled trials (RCTs) discussed in the review *that also* met Dr. Spector's inclusion criteria.

Premkumar 2019 was a systematic review of the effects of exposure to human-milk-based fortifiers[10] compared to cow's-milk-based fortifiers. (*Id*. at 12.) The review discussed only one RCT, O'Connor 2018, and it did not report a difference in the risk of NEC between the group exposed to human-milk fortifier compared to the group exposed to cow's-milk fortifier. Because

[10] As explained in the court's prior order, fortifiers are cow's-milk-based products produced for the purpose of adding key nutrients to an existing human milk (either mother's milk or donor milk) supply; though it shares the same ingredients as CMBF, unlike formula, it is not intended to substitute for or replace human milk. (*See* General Causation Order at 39 n.30.)

there was just one RCT study identified in Premkumar 2019, however, Dr. Hedges could perform no further meta-analysis.

Miller 2018 was a systematic review of human milk feeding and various morbidities, including NEC, in low birthweight infants. (*Id*. at 13.) The review examined 56 different studies, incorporating 44 in a meta-analysis. For Dr. Hedges' purposes, however, the review concluded, based on four RCT studies, that exposure to a higher dose of human milk relative to formula reduced the risk of NEC. These four studies–Corpeleijn 2016, O'Connor 2016, Schanler 2005, and Sullivan 2010—were the ones included in Dr. Spector's analysis, and Dr. Hedges performed a meta-analysis of the association between greater-CMBF diets and NEC observed in the four studies. (*Id*.) His Knapp-Hartung meta-analysis produced a relative risk of 2.25 (that is, a +125% increased risk of NEC from greater-CMBF diets)[11] with a wide confidence interval of 0.65–7.76, a *Q*-statistic of 7.41 (not significant), a large absolute heterogeneity ($\tau = .617$), and a moderate relative heterogeneity of $I^2 = 46\%$. Dr. Hedges interpreted these results as signifying an average relative risk of NEC that was "not statistically significant" with wide confidence intervals and "considerable" absolute and relative heterogeneity. (*Id*.)[12]

The Quigley 2019 review was a systematic review of the effects of CMBF compared to donor breast milk on preterm infants. (*Id*.) The review found nine randomized or "quasi-randomized" (Dr. Hedges does not explain this term) studies that suggested a higher risk of NEC in groups fed CMBF compared to groups of infants fed donor breast milk. (*Id*.) Dr. Hedges conducted a meta-analysis of three of these RCTs—Corpeleijn 2016, Cristofalo 2013, and

[11] For context, NEC occurs in between 4.6% and 10.4% of preterm infants. (Smith Rep. [613-7] at.)

[12] As discussed in more detail later in this order, Dr. Hedges later admits to having misinterpreted the results of Sullivan 2010—using a risk ratio of 1.01 (no difference between human milk and CMBF groups) when in fact the study observed a more significant association between CMBF and NEC outcomes. (Hedges Dep. Tr. [612-4] at 196:23–197:12.) As Dr. Hedges admits, this error likely resulted in a undercalculation of the risk ratio between CMBF and NEC. (*Id*. at 197:22–198:2.)

O'Connor 2016—that were included in Quigley 2019 and also in Dr. Spector's report. (*Id*.) His analysis produced a risk ratio of 2.24 (+124% increased risk of NEC from CMBF), a confidence interval from 0.20–25.55, a *Q*-statistic of 5.89 (not significant), a large absolute heterogeneity ($\tau = 0.824$), and a high I^2 value of 64%. (*Id*. at 13–14.) According to Dr. Hedges, these results, too, show an insignificant relative risk with a wide confidence interval and considerable absolute and relative heterogeneity. (*Id*. at 14.)

Finally, Quigley 2024 was a systematic review studying the effects of donor milk compared to CMBF in preventing NEC in preterm infants. (*Id*.) The review identified relevant RCTs and supported the conclusion that human donor milk reduces the risk of NEC compared to CMBF. (*Id*.) Dr. Hedges carried out a meta-analysis of five of these studies—Colaizy 2024, Corpeleijn 2016, Cristofalo 2013, Mills 2024, and O'Connor 2016—that were also included in Dr. Spector's report. The meta-analysis produced an average risk ratio of 1.80 (+80% risk of NEC in CMBF groups) with a confidence interval from .59 to 5.50, a *Q*-statistic of 8.47 (not significant), a large absolute heterogeneity ($\tau = 0.534$), and an I^2 value of 53%. (*Id*.) On these results, Dr. Hedges concluded that, consistent with his analysis of other reviews, the relative risk was insignificant, with wide confidence intervals and considerable absolute and relative heterogeneity. (*Id*.)

c. Analysis of Dr. Spector's Report

As noted, Dr. Hedges was asked to review the analysis and findings made by Plaintiffs' causation expert, Dr. Spector. Dr. Spector's report, as explained in the court's prior order, discusses three different categories of epidemiological studies testing the effect of CMBF on NEC outcomes: random control trials, cohort studies, and case control studies.[13] (*See* General Causation Order at 3.) Dr. Spector relied on separate meta-analyses (performed by Plaintiffs'

[13] An explanation of the differences between these study designs is explained in the court's General Causation Order. (*See* General Causation Order [646] at 3 n.3.)

biostatistician expert Dr. Rebecca Betensky) for each category of study in reaching his conclusion that a causal relationship exists between CMBF and NEC. (*Id*. at 7–8.) From ten random control trials observing Bell Stage 2 NEC or higher,[14] Dr. Betensky's meta-analysis observed a relative risk of 1.60 (+60% risk of NEC) in the CMBF-fed groups; from ten cohort studies meeting Dr. Spector's inclusion criteria, Dr. Betensky's meta-analysis observed a relative risk of 3.26 (+226% risk of NEC) in the CMBF-fed group; from four eligible case control studies, Dr. Betensky computed an odds ratio of 2.35 (+135% odds of NEC) in the CMBF-fed groups. (*Id*. at 8.) Looking at the same studies evaluated by Dr. Spector, Dr. Hedges performed separate meta-analyses of random control trials, cohort studies, and case control studies.

Dr. Hedges' meta-analysis of the ten random control trials (using the method described above) produced a similar average risk ratio—1.61 with a confidence interval between .96 and 2.68—as Dr. Betensky's meta-analysis. (Hedges Rep. at 16.)[15] Unlike Dr. Spector, however, Dr. Hedges determined that this increased risk was "not statistically significant." (*Id*.) His analysis of the heterogeneity between the ten studies revealed an insignificant *Q*-statistic (12.71), an insignificant absolute heterogeneity ($\tau = 0.332$), and a low I^2 of 30%. (*Id*. at 16–17.)[16] It also

[14] Bells Stage 2 refers to an advanced diagnosis of NEC where NEC is not only suspected (Bells Stage 1) but definitive and advanced. Stage 2 is also referred to as "confirmed NEC." NIH NICHD, Necrotizing Enterocolitis (NEC) Fact Sheet, https://www.nichd.nih.gov/health/topics/factsheets/nec (last accessed August 7, 2025).

[15] Because Sullivan 2010 was one of the studies included in this meta-analysis, Dr. Hedges' miscalculation of the risk ratio in Sullivan 2010 means that this value is also an undercalculation of risk.

[16] Dr. Hedges' heterogeneity analysis of these results does not indicate significant absolute (τ) or relative (I^2) heterogeneity; as he explains, even measures of heterogeneity can have uncertainty. That is, while the data is most consistent with low significance τ and I^2 values, Dr. Hedges also provides confidence intervals for relative and absolute heterogeneity that could include higher, more significant absolute and relative heterogeneity. (Hedges Rep. at 17.) The court, however, focuses on the calculated τ and I^2 values (which have the strongest support by the data), rather than the margins of the interval range.

revealed a wide prediction interval between 0.65 and 3.95—meaning that a new study consistent with Dr. Spector's selected RCTs could generate a vast range of results. (*Id*.)

Like Dr. Spector, Dr. Hedges conducted a meta-analysis of the five random control trials (included in the ten) that specifically compared a CMBF diet to a diet containing human-milk fortified human-milk based product called "Prolacta." (*Id*. at 18.) Dr. Hedges' meta-analysis of these studies produced a risk ratio of 1.86 (+86% increased risk of NEC in CMBF group)[17] with a confidence interval from .88 to 3.94. (*Id*.) In his heterogeneity analysis of these results, Dr. Hedges found insignificant *Q*-statistic, absolute heterogeneity, and relative heterogeneity values. (*Id*. at 18-19.) Again, however, he found a wide prediction interval, meaning that a new study could find a risk ratio as low as 0.71 or as high as 4.86. (*Id*. at 19.) In conducting this meta-analysis, Dr. Hedges observed that the five RCTs *not* included (*i.e.,* the non-Prolacta studies) involved studies where infants in both arms of the study were fed some amount of CMBF. (*Id*. at 20.) Performing a separate meta-analysis of the five non-Prolacta studies (which Dr. Spector and Dr. Betensky did not do), Dr. Hedges found a relative risk of 1.42—a 42% increase in risk of NEC in infants in the higher-CMBF groups. (*Id*. at 20.) Comparing the relative risk of the non-Prolacta studies (1.42) to that of the studies comparing CMBF-fed infants with those fed only human-milk and Prolacta (1.86), Dr. Hedges observed that the relative risk of NEC was actually lower for infants in the non-Prolacta studies (thus, those that received some CMBF in both groups), but that the difference was not statistically significant. (*Id*.)[18]

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[17] This is another meta-analysis relying on Dr. Hedges' miscalculation of the risk observed in Sullivan 2010.

[18] This finding is significant for Dr. Hedges' report because, in the non-Prolacta studies, infants in both arms of the study were given some amount of CMBF. In contrast, the infants in the Prolacta studies were either fed predominantly CMBF (the CMBF-arm) or no CMBF at all (the Prolacta arm). Thus, there was a greater difference in exposure to CMBF in the Prolacta studies compared to the non-Prolacta studies. (Hedges Rep. at 20.) As Dr. Hedges notes, if CMBF causes NEC, then studies with greater differences in exposure should exhibit higher relative risks of NEC than studies with lower differences in exposure—but Dr. Hedges observed no significant difference in risk of NEC between Prolacta and non-Prolacta studies. (*Id*.)

Dr. Hedges then performed a meta-analysis using the ten cohort studies. His meta-analysis produced an average risk ratio of 3.26 (identical to Dr. Betensky's meta-analysis) with a confidence interval from 2.29 to 4.66. (*Id*. at 21.) Noting the significant difference between this average risk ratio (3.26) and the ratio produced by the meta-analysis of the RCTs (1.60), Dr. Hedges notes that "this suggests that the two sets of studies are not estimating the same average effects." (*Id*.) His heterogeneity analysis of the cohort studies produced insignificant *Q*-test, absolute heterogeneity, and relative heterogeneity values, indicating low heterogeneity between the studies. (*Id*. at 21–22.) This was also reflected in his calculation of the prediction interval, which mirrored the confidence interval of 2.29 to 4.66. (*Id*. at 22.)

Finally, Dr. Hedges carried out a meta-analysis of the four eligible case-control studies included in Dr. Spector's report. The odds ratio he calculated is identical to the ratio calculated by Dr. Betensky (2.35) with a confidence interval from 1.17 to 4.72. (*Id*. at 23.) His heterogeneity analysis produced insignificant *Q*-test, absolute heterogeneity, and relative heterogeneity results, and the prediction interval matched the confidence interval—all indicative of low heterogeneity between the eligible case control studies. (*Id*.)

d. Assessment of Causality

In determining whether the results of the meta-analyses support a finding of causality, Dr. Hedges utilizes the Bradford-Hill criteria, a set of nine factors used in epidemiology to determine whether an observed association is suggestive of causation. (*Id*. at 24.)[19] Dr. Hedges relies mainly on his analysis of RCTs, which present the most reliable experimental evidence given the

[19] The criteria are generally articulated as (1) the strength of the association, (2) consistency of the exposure-disease relationship across different studies, (3) specificity of the association (whether the exposure is associated with only one disease or multiple outcomes), (4) the temporal relationship between exposure and disease, (5) "biological gradient," also known as dose-response (the amount of exposure resulting in the incidence of disease), (6) extent to which association has been established by experiment (as opposed to observation), (7) biological plausibility, (8) coherence with knowledge available to the researcher, and (9) analogy to similar causal mechanisms. (General Causation Order at 9); *see* Fed. Jud. Ctr., Reference Manual on Scientific Evidence 597–600 (3d. ed. 2011).

ability to control for bias and confounding factors in RCTs but not in cohort or case control studies. (*Id.*)

Beginning with the strength-of-association factor of the Bradford Hill analysis, Dr. Hedges explains that the average risk ratio observed in the RCTs (1.61) was not statistically significant, opining (without additional citation) that a doubling or tripling of risk is "usually expected if the association is causal." (*Id.*) Even the risk ratio of the Prolacta-studies (1.86) does not reach this doubling or tripling threshold that would support a finding of causality based on strength. (*Id.*)

Turning to the consistency factor, which looks at whether repeated experimentation observes a similar association (i.e. heterogeneity), Dr. Hedges views the results of the analyzed RCTs as "not particularly consistent." (*Id.* at 25.) *Q*-test and relative measures of heterogeneity were not significant in Dr. Hedges' calculations, but he observes that the absolute magnitude of heterogeneity ($\tau = 0.332$) in the RCTs was "not small" and that "moderate to substantial heterogeneity (inconsistency) of findings cannot be ruled out." (*Id.*)

Dr. Hedges then discusses the exposure response gradient (also known as the biological gradient factor or dose response), noting that the studies provided limited evidence about this factor. He notes, however, that comparing the RCTs involving high differences in exposure (i.e. the Prolacta studies) to the RCTs involving smaller differences in exposure (the non-Prolacta studies) did not produce a significant difference in average risk (1.86 in the former compared to 1.42 in the latter). (*Id.*) If there were a dose-response relationship between CMBF and NEC, Dr. Hedges reasons, the difference between Prolacta and non-Prolacta studies should have been greater.

Dr. Hedges does not discuss any additional Bradford Hill factors, beyond simply stating that "[n]o other Bradford Hill criteria point strongly to causation of NEC by formula." (*Id.*) Instead, Dr. Hedges concludes that based on his meta-analyses and review of previous studies, "to a reasonable degree of scientific certainty," exposure to CMBF does not cause NEC. (*Id.*)

B. Motion to Exclude Dr. Hedges

Plaintiffs raise two distinct challenges to Dr. Hedges report: one aimed at a specific miscalculation in his meta-analyses (*see supra* n.12), another targeted more generally at an alleged lack of "data underlying his analysis." (Pls.' Omnibus [612] at 4.)

1. Calculation Error

Plaintiffs argue that Dr. Hedges must be excluded because his report commits a significant calculation error that he admits to but has yet to correct by way of a supplemental report. (*Id*. at 3.) Specifically, in performing his meta-analyses, Dr. Hedges included an erroneous risk ratio for the Sullivan 2010 study. (*Id*.) Sullivan 2010 was an RCT comparing a human-milk diet fortified with cow's-milk-fortifier to two full human-milk diets fortified (at different intervals) with human-milk-derived fortifier (Prolacta). (*See* Sullivan 2010 [612-3] at 562–63.) Dr. Hedges' error resulted from the fact that Sullivan 2010 does not report its study outcomes in terms of risk ratio—those outcomes must be interpreted before they can properly be considered in the meta-analysis. (*See id*. at 565.) Sullivan 2010 does, however, find "a reduction in NEC of 50% and in surgical NEC of almost 90% in infants fed an exclusive human milk diet compared with a diet containing bovine milk-based products." (*Id*.) Dr. Betensky (Plaintiffs' expert) and Dr. Makuch (Abbott's expert) both interpreted Sullivan 2010 as reporting a risk ratio of 2.75. (*See* Betensky Rep. [616-38] at 56; Makuch Rep. ¶ 46.) For reasons that are unclear in the record, Dr. Hedges reported the risk ratio in Sullivan 2010 to be 1.01 (no difference in risk), a result he later admitted was "inconsistent" with the authors' stated finding of 50% or 90% reduced risk of NEC. (Hedges Dep. Tr. at 196:23–197:12.) He further acknowledged that using a risk ratio of 2.75 for Sullivan 2010, as Dr. Betensky and Dr. Makuch did, would likely move his results in favor of a stronger association between CMBF and NEC. (*Id*. at 197:22–198:2.)

Dr. Hedges' miscalculation likely resulted in an underreporting of the risk observed in Sullivan 2010, but the court is careful not to overstate the impact of this error. Even with this

misinterpretation of Sullivan 2010, Dr. Hedges' meta-analysis reached results identical to those of Dr. Betensky: Dr. Hedges found a 1.61 aggregate risk ratio from the 10 RCTs selected by Dr. Spector reporting Bell's Stage 2 or higher, Dr. Betensky found a risk ratio of 1.60 over the same data set. *See supra* p. 10.[20] The key difference between Dr. Hedges' opinion and that of Dr. Spector is the experts' interpretations of the (nearly identical) results. Dr. Spector concludes that an increased risk of 60% in CMBF-fed groups was statistically significant, while Dr. Hedges concludes that such a risk is not statistically significant. Dr. Hedges' causality analysis and application of the Bradford Hill criteria relies mainly on this identical risk ratio finding and does not appear to be impacted by his error in reading Sullivan 2010. Furthermore, Dr. Hedges' meta-analyses of Premkumar 2019, Quigley 2019, or Quigley 2024 (none of which included Sullivan 2010 in their reviews), were not impacted by the error. *See supra* pp. 7–9.

Nonetheless, Dr. Hedges has admitted to this error—or at least "inconsistency"—in his preparation of several meta-analyses but has not corrected or explained this inconsistency in an amended or supplemental report. Until he does so, the court will exclude testimony as to the results of any meta-analysis that rely on his erroneous interpretation of Sullivan 2010.[21] The court does not exclude, on this basis, testimony regarding his meta-analyses of existing reviews that do not include Sullivan 2010, his meta-analyses of Dr. Spector's selected observational studies, and Dr. Hedges' application of the Bradford Hill criteria. Of course, absent a supplemental report, Plaintiffs will be free to cross-examine Dr. Hedges about a known and unaddressed inconsistency in his report.

[20] Of course, this does not exclude the possibility that Dr. Hedges' method could have produced a more favorable meta-analysis to Plaintiffs than Plaintiffs' own experts.

[21] This would include Dr. Hedges' meta-analysis of the cases discussed in the Miller 2018 review, of the 10 RCTs selected by Dr. Spector, and of the five Prolacta studies.

2. Lack of Replicable Method

Plaintiffs' broader challenge to Dr. Hedges is that his report omits mention of the specific software used to compute his meta-analyses and does not specify the exact data inputs Dr. Hedges used to include the various studies into his calculations. (Pls.' Omnibus at 4–5.) Absent these facts, Plaintiffs argue, Dr. Hedges' method is not replicable and is, therefore, not reliable.

"[E]xpert opinions that cannot be objectively replicated are subject to exclusion." *In re Paraquat Prods. Liab. Litig.*, 730 F. Supp. 3d 793 (S.D. Ill. 2024) (citing *Timm v. Goodyear Dunlop Tires N. Am., Ltd.*, 932 F.3d 986, 994 (7th Cir. 2019)). This means that "[a]n expert must offer good reason to think that . . . . [s]omeone else using the same data and methods [will] be able to replicate the result." *Zenith Elecs. Corp. v. WH-TV Broad. Corp.*, 395 F.3d 416, 419 (7th Cir. 2005). Plaintiffs contend that, because he failed to identify the specific software used to conduct his meta-analysis calculations, Dr. Hedges' report is not replicable. The court disagrees. The software used to execute a method is not the same as the method itself. Dr. Hedges' report makes clear that he used the Knapp-Hartung adjustment to the random effects model and clearly explains the kinds of calculations he made in his heterogeneity analysis. *See supra* p. 5. In his deposition, Dr. Hedges not only clarifies that he used the "Metafor" software to run these calculations, but further notes that "if you specify clearly enough the analysis that you did . . . it's not necessary to know which software was used because any software ought to give you the same answer, unless there is an error in the software." (Hedges Dep. Tr. at 42:12–16.) Plaintiffs do not appear to argue that the specific software used to run a meta-analysis changes the outcome given a specific method like Knapp-Hartung, nor do they suggest that the Knapp-Hartung method is an ambiguous or problematic method. Absent such arguments, the omission of "Metafor" in his report does not justify excluding Dr. Hedges' opinions. Plaintiffs also argue that Dr. Hedges' method is not replicable because his report does not state what information he specifically took from the various studies and input into his meta-analysis model to reach his results, or the "input data." (Pls.' Omnibus at 4–5.) But Dr. Hedges provided this information in

his deposition, explaining that from each RCT, he input the sample size (number of infants) of each group and the number of events (that is, incidence of NEC) in that group. (*See* Hedges Dep. Tr. at 43:17–25.) He further testified that such an approach is consistent with what any "good statistician" or "competent researcher" would do. (*Id*. at 44:4–45:1.) Again, Plaintiffs do not challenge this approach but appear to focus on the absence of this information from the text of the original report. That information is missing from an expert report may be relevant to an objection to proper disclosure under Federal Rule of Civil Procedure 26(a)(2), but a district court may rely on an expert's subsequent deposition testimony to determine whether the expert's methods are reliable. *See Lapsley v. Xtek, Inc.*, 689 F.3d 802, 814 (7th Cir. 2012) (affirming district court's consideration of expert's "report, calculations, and deposition testimony available" at the time the court ruled on the opposing party's *Daubert* motion). Here, Dr. Hedges' deposition testimony removes all ambiguity as to the specific inputs used in his meta-analysis.

Except as noted above, Plaintiffs' *Daubert* challenge to Dr. Hedges is overruled.

II. Dr. Ericka Claud

Dr. Claud is a neonatologist and pediatrician engaged by Mead Johnson to respond to the report written by Plaintiff expert Dr. Jennifer Sucre; her testimony is expected to be offered in the *Inman* bellwether.

A. Dr. Claud's Report

1. Qualifications

Dr. Claud is a board-certified neonatologist and Tenured Professor of Pediatrics at the University of Chicago. (Claud Rep. [612-6] at 3.) She received her M.D. and completed her pediatric residency in 1990 and 1993, respectively, both at Northwestern University. (*Id*.) After residency, Dr. Claud worked as an Instructor of Clinical Pediatrics at Northwestern before completing a fellowship in neonatology at Northwestern's Children's Memorial Hospital. (*Id*.) She then joined Harvard Medical School, holding a clinical position at Boston Children's Hospital and

completing a research fellowship at Massachusetts General Hospital. (*Id*.) She joined the University of Chicago faculty in 2004. (*Id*.)

Dr. Claud's research focuses on the development and mechanism of NEC. She has received several NIH grants to investigate the role of microbes in the development of preterm intestines and has published numerous articles relating to the development and treatment of NEC in preterm infants. (*Id*. at 3–4.)

2. Methodology

Mead Johnson retained Dr. Claud to conduct a literature review and provide an opinion as to whether CMBF plays a causal role in the development of NEC in preterm infants. (*Id*. at 4.) Her report does not explain her method for selecting relevant literature, nor does it detail a process for reviewing the literature to arrive conclusions. But the report contains an appendix listing each publication that she reviewed in preparing her opinions. (*See id*. at 31–42.)

Dr. Claud's report begins by providing background information on the stages of fetal development and preterm birth, about how preterm infants are treated and fed (using mother's milk, donor milk, and formula) in NICUs, the intestinal structure of preterm infants, and how NEC can present at the early stages of life. (*Id*. at 4–12.) Dr. Claud then explains how prematurity, particularly intestinal immaturity that results in bacterial colonization in the intestines and altered blood flow through the intestines, is the primary risk factor for the development of NEC. (*Id*. at 13–18.) Next, Dr. Claud opines that mother's milk *reduces* the risk of NEC—in contrast with the notion that consumption of CMBF increases the risk. In support, Dr. Claud cites studies observing lower incidence of NEC in higher-mother's milk diets, and explains that mother's milk contains antibodies, enzymes, and stem cells that help with digestion and repairing the epithelial wall. (*Id*. at 19.) Indeed, Dr. Claud notes that even donor milk, because it is pasteurized, does not contain all these ingredients, which explains the higher incidence of NEC in infants fed donor milk when compared to infants fed mother's own milk. (*Id*.) CMBF too, Dr. Claud notes, does not contain

the ingredients that mother's milk possesses in protecting against NEC, but serves the crucial purpose of providing necessary nutrients to preterm infants. (*Id*. at 20.)

Dr. Claud then turns to a review of Dr. Jennifer Sucre's report. Dr. Sucre is Plaintiffs' neonatology expert who opines, relying on *in vivo* animal studies, that carbohydrates, proteins, and fats in CMBF are malabsorbed by the preterm gut and cause NEC by feeding bacteria and triggering the TLR4 receptor. (*See* General Causation Order [646] at 16–17.) Dr. Claud disagrees with each aspect of Dr. Sucre's theory of causation. Beginning with Dr. Sucre's theory that the carbohydrates in CMBF are malabsorbed by pre-term infants, Dr. Claud explains that Dr. Sucre "misreads the animal literature." (*Id*. at 23.) Specifically, Dr. Sucre relied on animal studies showing poor digestion of maltodextrin (a carbohydrate found in CMBF) associated with NEC-like injuries in preterm piglets; according to Dr. Claud, this reliance is inappropriate because the enzymatic profile of piglets at birth is the opposite of human infants. (*Id*. at 23.) That is, the intestines in preterm human infants have low amounts of lactase (the enzyme that breaks down lactose) and high amounts of maltase (the enzyme that breaks down maltose and maltodextrin) at birth. Piglets instead have high lactase and low maltase in the preterm period—thus, findings of piglets' inability to digest maltodextrin and other glucose polymers cannot be extended to preterm infants. (*Id*.) If anything, Dr. Claud explains, the piglet studies support a finding that it is the inability to digest lactose (found in human milk and CMBF alike) that leads to NEC, not the glucose polymers specific to CMBF. (*Id*.)

Dr. Claud disagrees, further, with Dr. Sucre's theory that the protein composition of CMBF (containing more casein proteins than whey proteins relative to human milk) makes it more likely to be malabsorbed by preterm infants. According to Dr. Claud, "Dr. Sucre is wrong about the protein composition of both preterm formula and human milk." (*Id*. at 24.) Dr. Sucre's report asserts that human milk contains a 60:40 ratio of casein (harder to digest) to whey proteins (easier to digest), while bovine milk contains an 80:20 casein to whey ratio—meaning CMBF has higher ratios of harder-to-digest proteins. (*See* Sucre Rep. [616-35] at 27.) But Dr. Claud explains that

in comparison to the milk produced by mothers of full-term infants, preterm human milk tends to have a much higher ratio of whey to casein proteins,[22] and that (due to concerns about maldigestion) preterm formula contains a high ratio of whey protein to "mimic the ratio in early human milk." (Claud Rep. at 24–25.) Indeed, Dr. Claud is not aware of *any* preterm formula with an 80:20 casein to whey ratio. (*Id*.) Further, Dr. Claud disagrees with Dr. Sucre's understanding of the literature relating to preterm digestion of proteins. Dr. Sucre would testify that human milk contains bioactive ingredients that made it easier digest than CMBF. Dr. Claud agrees that this is true but asserts that the literature in fact supports the conclusion that mother's-own-milk is easier to digest than both CMBF *or* donor milk, as the crucial bioactive ingredients are denatured in the pasteurization process. (*Id*. at 25.)

Finally, Dr. Claud refutes Dr. Sucre's conclusion that the fats in CMBF cause or contribute to the development of NEC. Dr. Sucre's conclusion regarding fats relies on the fact that CMBF does not contain the lipase enzyme found in human milk, which aids in the digestion of fats. (*See* Sucre Rep. at 28.) She further relies on mice studies showing that undigested fats lead to NEC in preterm subjects. (*Id*.) Dr. Claud does not dispute that CMBF does not include the lipase enzyme found in mother's milk but disagrees with Dr. Sucre's reliance on mice studies showing an association between undigested fats and NEC. (Claud Rep. at 27.) In Dr. Claud's view, the mice studies referenced by Dr. Sucre are inapplicable to human infants for three reasons: first, the mice in the studies were fed a formula containing twice as much fat content as found in commercial preterm formula; second, NEC did not occur in the studies unless the study authors *induced* hypoxia (removed oxygen) and *implanted* the mice with NEC bacteria; and third, the mice studies reference other studies finding *decreased* rates of NEC with the inclusion of long-chain fatty acids (like those found in CMBF) into the diet. (*Id*. at 27–28.)

[22] As Dr. Claud explains, the protein ratios of human milk vary at different stages of an infant's maturity. "Mature" human milk is 60:40 whey to casein, while preterm human milk tends to vary between 70:30 and 90:10 whey to casein. (Claud Rep. at 24.)

Dr. Claud raised other concerns about Dr. Sucre's conclusions regarding fats, as well. For one, she again points out that the purported "difference" between mother's milk and CMBF for digesting fats—the presence of lipase—applies to donor milk and CMBF alike; the process of pasteurizing donor milk destroys or denatures lipase. (*Id*. at 29.) Dr. Claud further reasons that Dr. Sucre contradicts herself by asserting that CMBF results in "unbound free fatty acids" leading to NEC: to the contrary, Dr. Claud asserts, the digestion of fats *releases* fatty acids. (*Id*.) Thus, the fact that mother's milk is easier to digest than CMBF means that infants fed human milk will release *more* fatty acids than those consuming CMBF, contrary to Dr. Sucre's conclusion. (*Id*.) Finally, she explains that certain long-chain fatty acids contained in CMBF are required for preterm brain development and must be included in the preterm diet (via CMBF or other mechanisms) post-delivery. (*Id*.)

Dr. Claud concludes her report by opining that the pathogenesis of NEC is "unclear" but "likely related to the combination of bacterial colonization and enteral feeding . . . combined with a premature intestine that is immature." (*Id*. at 30.) She further opines that "[m]other's own milk is protective against NEC because it enhances maturation of the intestine" and that "there is nothing about the macronutrients in formula suggesting that it causes or contributes to cause the development of NEC." (*Id*.)

B. Motion to Exclude Dr. Claud

Plaintiffs argue that Dr. Claud's opinion must be excluded for two reasons. First, they argue that her report fails to articulate a methodology for her search and analysis of relevant literature. (Pls.' Omnibus at 6–7.) Second, they argue that Dr. Claud's report is not reliable because it excludes literature that Plaintiffs contend is "contrary evidence" to her conclusions. (*Id*. at 7.)

1. Lack of Methodology

As explained *supra* p. 18, Dr. Claud's report lists the articles she reviewed before arriving at her conclusions but does not describe the method used to search for those articles, nor a

specific analytical method for evaluating and weighing the information from each article. In her deposition, Dr. Claud confirmed that she did not apply a "a specific standardized methodology," but rather "wrote [her] opinion based on [her] expertise and decades of research in this field and clinical work supported by specific literature." (Claud Dep. Tr. [612-7] at 44:8–12.) Plaintiffs argue that this is fatal for the admission of her testimony under *Daubert*.

Daubert and Federal Rule of Evidence 702 require that an expert's testimony "rests on a reliable foundation." *Krik v. Exxon Mobil Corp.*, 870 F.3d 669, 674 (7th Cir. 2017). Neither *Daubert* nor Seventh Circuit precedent require that an expert name a "specific standardized methodology" in reaching her conclusions; *Daubert* directs the district court to determine "whether *the reasoning or* methodology underlying the testimony is scientifically valid." *Daubert*, 509 U.S. at 592–93 (emphasis added). "The critical inquiry is whether there is a connection between the data employed and the opinion offered," and "it is the opinion connected to existing data 'only by the *ipse dixit* of the expert' [] that is properly excluded under Rule 702." *Manpower, Inc. v. Ins. Co. of Pennsylvania*, 732 F.3d 796, 806 (7th Cir. 2013), citing *General Electric Co. v. Joiner,* 522 U.S. 136, 146 (1997).

While Dr. Claud does not articulate a specific methodology like the Knapp-Hartung meta-analysis method or a formal systematic review method, the opinions in her report are far from *ipse dixit*. Nearly every assertion in her report is followed by a citation to the scientific literature, which Plaintiffs do not dispute represent peer-reviewed and sound methodologies. Moreover, as Mead Johnson points out, the opinions contained in Dr. Claud's report are consistent with her prior publications regarding the biological mechanisms of NEC—publications that have been subject to peer review and have achieved general acceptance in the field of neonatology. (*See* Mead Johnson Resp. [622] at 3–4.) Her theories, therefore, meet the indicia for reliability that *Daubert* requires. *See Baugh v. Cuprum S.A. de C.V.*, 845 F.3d 838, 844 (7th Cir. 2017) ("When determining the reliability of a qualified expert's testimony under *Daubert*, courts are to consider . . . whether the theory has been subjected to peer review [and] whether the theory has been

accepted in the relevant scientific community."). Given Dr. Claud's extensive expertise in the study of NEC and the consistency between her prior publications and her expert report, the court finds that her opinion is sufficiently reliable under *Daubert*, notwithstanding the absence of a specified methodology.

2. Failure to Consider Contrary Evidence

Plaintiffs' alternative objection to Dr. Claud's testimony is that she refused to consider evidence in support of a causal association between CMBF and NEC and relied on sources in her report that she "disavowed" in her academic work. (Pls.' Omnibus at 7–8.)

In support of the first contention, that Dr. Claud ignored contrary evidence, they point to a specific article (*see* Johnson 2015 [612-8]) that found a statistically significant increase in NEC in infants fed any amount of CMBF and proposed (citing other sources) a biological mechanism by which CMBF damaged the epithelial lining and caused NEC. (Pls.' Omnibus at 8; *see* Johnson 2015 at 275.) In her deposition, Dr. Claud admitted that the article was not cited as a reference in her report, and that she did not review the various studies cited in Johnson 2015 to support the theory that CMBF causes NEC. (*See* Claud Dep. Tr. at 158:12–162:19.) Dr. Claud did, however, state familiarity with at least some of these studies from her work and general knowledge in the field. (*Id*. at 162:9–14.) Indeed, Dr. Claud has previously discussed two studies of particular importance cited in the Johnson 2015 article, Taylor 2009 and Penn 2012, in her prior academic work. (*See* Mead Johnson Ex. 7 [622-7] at 18.)

Certainly, "[e]xperts who engage in cherry-picking of the evidence fail to satisfy the scientific method and *Daubert*." *Van v. Ford Motor Co*., 332 F.R.D. 249, 269 (N.D. Ill. 2019) (citing *Barber v. United Airlines, Inc*., 17 F. App'x 433, 437 (7th Cir. 2001). Had Dr. Claud systematically ignored research that directly contradicted her conclusions, her methods and opinions would not be reliable. But Dr. Claud's omission of potentially relevant studies (many of which she had previously reviewed) does not, in the court's view, rise to the level of cherry-picking, or otherwise cast doubt on the reliability of her opinions. For one, there is at least some dispute as to whether

the studies mentioned by Plaintiffs are, in fact, inconsistent with her theories. (*See* Mead Johnson Resp. at 6; Claud Dep. Tr. at 265:21–266:7.) More crucially, the court is mindful of the kind of opinion Dr. Claud is offering for these cases. Unlike Dr. Sucre, Dr. Claud was not tasked with performing a formal literature review on a question outside the scope of her prior research; rather, her report presents her own understanding of the mechanisms of NEC based on her extensive prior research on the subject. (*See* Claud Rep. at 3–4.) *Daubert* is a "flexible" standard meant to "be geared toward the precise sort of testimony at issue." *Gopalratnam*, 877 F.3d at 780 (quoting *Lees v. Carthage Coll.*, 714 F.3d 516, 521 (7th Cir. 2013)). Dr. Claud's precise testimony is a summation of her experience in the field and her informed understanding of prior research; the report provides sufficient support for this opinion with clear reasoning and citations to the relevant literature.

As for the argument that Dr. Claud relied on sources in her report that she disavowed in prior academic work, Plaintiffs overstate the conflict between her report and prior writings. Plaintiffs note, first, that in her report, Dr. Claud writes that "hydrolysis increases the osmolality of formula, which itself has raised concerns for an increased risk of NEC."[23] (Claud Rep. at 26.) A 2019 textbook co-authored by Dr. Claud, however, states that "[h]igh osmolar formulas were also once thought to put a strain on the GI tract of the immature preterm infant . . . . This may not be true because [of] recent research." (*See* Claud Dep. Tr. at 166:19–167:4.) There is clearly some tension between these two statements; her report notes the concern that osmolar formula increases the risk of NEC while her textbook notes that new research has cast doubt on this concern. But a recognition that new research raises questions about a previous theory is not equivalent to a "disavowal" of that theory. Plaintiffs may explore this tension on cross-

[23] As Dr. Claud explains, osmolarity and osmolality are two ways of expressing the concentration of formula in a given solute—osmolarity measures the concentration in terms of volume (liters), osmolality in terms of weight (kilograms). (Claud Dep. Tr. at 167:17–22.)

examination, but the court does not find that this minor conflict, on a peripheral aspect of Dr. Claud's opinion, provides a basis for questioning the reliability of Dr. Claud's entire report.

The motion to bar her testimony is denied.

III. Dr. Robert Makuch

Dr. Makuch is a biostatistician engaged by Abbott to respond to the meta-analysis performed and analyzed by Plaintiffs' experts Dr. Spector and Dr. Betensky; his testimony is expected to be offered in the *Etienne* bellwether trial.

A. Dr. Makuch's Report

1. Qualifications

Dr. Makuch is a Professor Emeritus of Biostatistics at Yale University, where he has served as Professor (first as Associate Professor, then tenured Full Professor) in the Department of Biostatistics since 1986. (Makuch Rep. [612-9] ¶ 6.) He received a B.A. in Mathematics from the University of Connecticut in 1972, an M.A. in Mathematics from the University of Washington in 1974, and his Ph.D. from Yale in 1977. (*Id*. ¶ 4.) Between 1977 and 1986, Dr. Makuch held positions at the National Cancer Institute within The National Institutes of Health. (*Id*. ¶ 5.) He has authored or co-authored over 215 peer-reviewed articles, "most of which relate to the design, conduct, analysis, and interpretation of clinical studies." (*Id*. ¶ 9.)

2. Methodology

Akin to the work performed by Dr. Hedges, Dr. Makuch's report is a systematic review and meta-analysis of epidemiological studies observing a relationship between CMBF and NEC in preterm infants. (*See id*. ¶ 26.) He describes his meta-analysis method as an eight-step process. The process begins with (1) developing a protocol and (2) generating a hypothesis to guide the meta-analysis. (*Id*. ¶ 24.) This is followed by (3) a literature search strategy that results in a selection of studies followed by (4) a methodological quality assessment (i.e. testing for bias in results) and (5) data extraction. (*Id*.) After the data from the studies is extracted, he (6) evaluates the heterogeneity of the studies, (7) analyzes and presents the meta-analysis of the studies, and

(8) performs a subgroup and sensitivity analysis (a test of whether the result is robust by observing the effect of including or excluding small number of studies). (*Id.*)

a. Protocol Development and Hypothesis

Dr. Makuch begins his meta-analysis by defining his research question, or hypothesis, as "whether an association exists between cow's milk-derived preterm products and NEC in RCTs of premature or VLBW (very low birth weight) infants in which at least 75%[24] of the enteral feeds in the cow's milk group (on a group-wide basis) consisted of human milk." (*Id.* ¶ 26.) In defining his protocol, he created *a priori* inclusion criteria—requirements for studies that would be included in his analysis. (*Id.* ¶ 27.) The inclusion criteria were (1) the studies had to be RCTs, (2) the study subjects consisted of premature (<37 weeks gestational age) or very-low-birth-weight (<1500 grams) infants, (3) the studies compared CMBF to human milk or human milk-derived feeding products, (4) the studies assessed confirmed (as opposed to suspected) cases of NEC (Bell's Stage 2), and (5) the studies involved a diet of at least 75% human milk as a percentage of total enteral feeds in the group fed CMBF. (*Id.*) In determining whether a study met the "75%" criteria, Dr. Makuch accepted the percentages reported by the study authors if they were provided. (*Id.*) Otherwise, Dr. Makuch would estimate the percentage of human milk in the diet based on the mixing instructions for the specific CMBF-product in the study; for example, if the study involved Abbott's liquid cow's-milk fortifier, which comes with instructions for a 5 ml fortifier to 25 mL human-milk mixing ratio, Dr. Makuch assumed an 83% human-milk diet. (*Id.*) Dr. Makuch did not limit his analysis to cow's-milk formula, but included studies involving formula and

[24] Dr. Makuch's decision to focus on studies featuring diets of at least 75% human milk is based on his "understand[ing] that human milk comprised over 75% of the plaintiff's enteral feeds prior to NEC in this case." (Makuch Rep. ¶ 28.) By "plaintiff," he is referring to RaiLee Mar, the child of Plaintiff Ericka Mar in the *Mar* bellwether case, in which the court granted summary judgment to Abbott for reasons unrelated to Dr. Makuch's testimony. Dr. Makuch does not explain whether this approach is also appropriate for the *Etienne* bellwether, in which his testimony will also be offered.

cow's-milk-based fortifier, given the shared primary ingredients between the two categories of products. (*Id*. ¶ 30.)

b. Literature Search and Qualitative Assessment

With his hypothesis and *a priori* inclusion criteria established, Dr. Makuch designed a search of the database PubMed to produce relevant studies. He used the following search terms: "vlbw OR very low birth weight infants OR premature infants OR preterm infants OR NEC OR necrotizing enterocolitis OR necrotising enterocolitis." (*Id*. ¶ 31.) He further applied filters limiting the search results by article type (RCTs only), language (English only), and publication date (01/01/1970 to present). (*Id*.) The search yielded 6,624 studies. (*Id*.) He supplemented this search with studies cited in Cochrane systematic reviews,[25] referenced in published papers (he does not explain which), and articles cited in Plaintiffs' complaints and expert reports. (*Id*. ¶ 32.)

Dr. Makuch screened the titles and abstracts of publications that turned up in his search, presumably (Dr. Makuch does not state explicitly) excluding any study that did not appear to meet his *a priori* inclusion criteria. (*Id*. ¶ 33.) Of the 6,624 studies, 32 studies survived this initial screening; from there, Dr. Makuch proceeded to full-text review, where he further excluded studies that appeared (in their title and abstract) relevant but did not actually meet his criteria. (*Id*.) Ultimately, after applying his *a priori* inclusion criteria to a full-text review of the 32 studies, Dr. Makuch selected nine random control trials for inclusion in his meta-analysis. (*Id*. ¶ 34.)

Dr. Makuch's qualitative assessment of these nine studies involved assessing each study for selection bias, performance bias, detection bias, and attrition bias. (*Id*. ¶ 35.) Selection bias was determined by assessing whether the studies properly used random sequence generation

[25] In addition to providing a handbook on best practices for performing systematic reviews and meta-analyses, Cochrane, or the Cochrane Library, is also a database of systematic reviews. *See* Cochrane Library, https://www.cochranelibrary.com/cdsr/reviews. (last accessed August 8, 2025). Dr. Makuch lists four studies (Premkumar 2019, Brown 2020, Quigley 2019, and Quigley 2024) that he found by searching for systematic reviews in Cochrane, as well as the systematic reviews in which the studies were cited. (Makuch Rep. at 10 n. 22–24.) He does not explain what search terms or other methods he used to find these systematic reviews.

(groups assigned by random sequence) and allocation concealment (group assignment hidden from researchers). (*Id.*) Performance bias—bias resulting from one group receiving different treatment than the other—was tested by assessing whether there was proper blinding of participants and personnel. (*Id.*) Detection bias—bias resulting from one group being tested more than another—was evaluated by assessing the blinding of outcome assessments. (*Id.*) Attrition bias—bias resulting from one population disproportionately dropping out of the study—was determined by assessing the presence of incomplete outcome data. (*Id.*) Dr. Makuch rated each study from "low risk" to "high risk" on each one of these assessments—random sequence generation, allocation concealment, blinding of participants and personnel, blinding of outcome assessment, and incomplete outcome data—and presented these ratings on a table. (*See* Risk of Bias Assessment, Makuch Rep. at 59–61.) As many of the studies Dr. Makuch selected for review had been previously reviewed for these biases in a Cochrane-published review, Dr. Makuch also included the Cochrane rating for each bias assessment on the table. (*Id.*) While some studies, per Dr. Makuch's assessment, showed signs of "high risk" in one or more categories, Dr. Makuch concluded "based on his 40+ years of designing, conducting, analyzing, and interpreting RCTs" that there were "no biases of such seriousness to call into question the reliability of the results." (*Id.* ¶ 39.)

c. Data Extraction and Meta-Analysis

Dr. Makuch was able to extract data from all nine studies that passed his literature review and bias assessment. In doing so, Dr. Makuch relied on the "intention-to-treat" ("ITT") population reported by the RCT, which "includes all individuals randomized to that group whether or not they received the assigned intervention." (*Id.* ¶ 18.) This means that if the formula arm of a study began with 100 infants, lost 20 infants from the study prior to receiving formula, and reported 20 cases of confirmed NEC, Dr. Makuch would include that study as reporting a 20% incidence of

NEC (20/100) in the formula group, not 25% (20/80). Dr. Makuch explained, and materials he cited confirm, that "[a]nalyzing the ITT population is widely considered the gold standard." (*Id.*)[26]

Using the meta-analysis software ReviewManager, Dr. Makuch analyzed the data set under the fixed-effects Mantel-Haenzel method of meta-analysis—different than the random effects model utilized by Mead Johnson expert Dr. Hedges but identical to the method used by Plaintiffs' biostatistician Dr. Betensky. (*Id.* ¶ 41.) In combining the results of the nine eligible studies, Dr. Makuch calculated two values: risk ratio and risk difference. (*Id.* ¶ 43.) Risk ratio, as discussed in the reports of Dr. Hedges and Dr. Spector, is a numerical representation of the relative risk of developing a health outcome between two groups; if the average incidence of NEC in human-milk-fed groups was 10% and the average incidence of NEC in CMBF-fed groups was 15%, the risk ratio would be 1.50 representing a 50% increased risk. Risk difference, on the other hand, is not a measure of relative risk but of the delta in absolute risk between the two groups; given the same hypothetical incidence of NEC described above, the risk difference would be .05, or 5% (15% - 10%). (*Id.* ¶ 44.) Risk difference is useful as an alternative measure of risk involving low incidence outcomes like NEC, and otherwise can account for studies with zero events in one group. (*Id.* ¶ 45.)

Dr. Makuch's meta-analysis of the nine eligible RCTs resulted in an average risk ratio of 1.16 with a confidence interval from 0.79 to 1.71 (that is, +16% relative risk), and a risk difference of .01 (that is, +1% absolute risk)[27] with a confidence interval between -.01 to 0.03. (*Id.* ¶¶ 46–

[26] Dr. Makuch goes on to explain that looking at the intention-to-treat population is the gold standard because doing so "(i) preserves randomization, (ii) reflects real-world clinical scenarios, (iii) reduces bias (i.e., by not excluding non-compliant patients), and (iv) limits arbitrary subgroup analyses and issues of multiplicity." (Makuch Rep. ¶ 18.)

[27] It may be useful to put these findings in terms of the absolute risk of NEC for preterm infants fed CMBF (which can be determined for Dr. Makuch's meta-analysis because he calculates both relative risk and absolute risk difference). If CMBF increases the relative risk of NEC by 16%, and the absolute risk by 1%, then the court can calculate that this represents an increase of the risk of NEC from 6.25% to 7.25%—this is generally consistent with risk of NEC in preterm infants between 4.6% and 10.4%. (*See supra* n.11)

49.) Neither of these results is statistically significant. (*Id.*) Dr. Makuch does not discuss the heterogeneity of studies observed in the meta-analysis, though raw measures of heterogeneity (I^2, Chi^2, P value) are presented in tables in his report. (*See id.* ¶¶ 46, 48.)

Dr. Makuch also performed a sensitivity analysis by adding a tenth RCT (O'Connor 2016) that was slightly below the 75% human-milk-diet threshold to the data set and running the meta-analysis again. (*Id.* ¶ 50.) With O'Connor 2016 included in the set of studies, the risk ratio moved to 1.35 and risk difference moved to 0.02—values that Dr. Makuch opines remain not statistically significant and thus adding confidence in the robustness of the primary meta-analysis. (*Id.* ¶¶ 50–51.) Reporting these results, Dr. Makuch concludes that "meta-analytic evidence shows no evidence of a statistically significant association between cow's milk-derived preterm products and NEC" in RCTs involving pre-term infants and at least 75% human-milk diets. (*Id.* ¶ 52.)

d. Critique of Plaintiffs' Experts

Dr. Makuch concludes his report by explaining "[m]ethodological [d]efficiencies" in the reports of Dr. Spector and Dr. Betensky. His critique is only cursory, stating that "their analyses . . . suffer from flaws including but not limited to inclusion of observational studies and failure to include all RCTs using their own criteria." (*Id.* ¶ 53.) With respect to his "failure to include" concern, however, Dr. Makuch has not identified the RCTs that fit Dr. Spector's and Dr. Betensky's "criteria" but were missing. He further "take[s] issue with Dr. Spector's failure to identify a group of studies with sufficient homogeneity to combine," but does not explain why the studies that Dr. Spector did choose lacked sufficient homogeneity. (*Id.*)

B. Motion to Exclude Dr. Makuch

Plaintiffs seek to exclude Dr. Makuch for three reasons: (1) his requirement that eligible studies include diets of 75% human-milk or more renders his opinion inapplicable to the facts of *Etienne*, (2) he did not apply his inclusion criteria reliably, and (3) his methodology is not reproducible. (Pls.' Omnibus at 9.)

1. Applicability to Facts of *Etienne*

Prior to the court's grant of summary judgment in the *Mar* bellwether litigation, and in relation to this motion, the parties strenuously debated the proper way to measure an infant's exposure to CMBF. Abbott has argued that exposure to CMBF must be measured by taking the amount of CMBF ingested as a proportion of the infant's total feedings beginning from birth and until the onset of NEC. RaiLee Mar was given 72 feedings of 100% mother's milk and then three feedings of 50/50 human-milk/CMBF before being diagnosed with NEC—by Abbott's calculation, a 5% CMBF diet. (*See Mar* Summ. J. Order [96] in No. 22 C 232 at 5–6.) Under Abbott's definition of exposure, the relevant causation question in *Mar* was whether a 5% CMBF could cause NEC. In contrast, Plaintiffs have argued that exposure must be measured "in the days prior to the onset of NEC." (Pls.' Omnibus at 10.) RaiLee was transitioned from a 100% mother's-milk diet to a 50% CMBF diet immediately before developing NEC. Plaintiffs framed the operative question in *Mar* as whether a 50% CMBF diet could cause NEC. (*See Mar* Summ. J. Order at 5–6.) Because the court granted summary judgment on different grounds, it did not resolve the proper way to measure exposure to CMBF. (*See id.*)

Plaintiffs' motion to exclude Dr. Makuch brings this contested issue to a head. Dr. Makuch has "no opinion on [infants] in whom the human milk threshold was less than 75 percent," and reviewed no studies where a group of infants in any study received more than 30% formula. (Makuch Dep Tr. [612-11] at 51:15–18; 93:1–10.) The record does not include information about how much CMBF and human milk D.B. (the infant in *Etienne)* was fed while admitted to the NICU; but if the court were to include that the proper measure of D.B.'s intake of CMBF would show he received more than 30%, Dr. Makuch's meta-analysis may not help a jury determine whether CMBF did or did not have a causal effect. While Abbott's counsel has made forceful arguments in favor of their interpretation of exposure, Dr. Makuch's voice has been missing; his report provides no basis for his decision to limit his search for studies to those involving 75% human-milk diets as it applies to the *Etienne* case. The only justification he provides for limiting the scope

of his search to 75% human milk diets is a citation to a report prepared by Dr. Martin (Abbott's *Mar*-specific expert) asserting that RaiLee Mar's diet exceeded 75% human milk across all enteral feedings. (Makuch Rep. ¶ 28 n.17.) Given the recognized significance of this issue, the court would expect Dr. Makuch to explain his rationale for considering the percentage of formula across all enteral feedings as the relevant measure of exposure. Relatedly, he has not yet explained why studies that test higher percentages of formula diets (but may map on to specific feeding windows within the infant's time in the NICU) are so irrelevant as to justify exclusion from his analysis. He further provides no mention of D.B.'s feeding regimen that explains why the 75% threshold is applicable to his case.[28] Ultimately, it is the party seeking to enter expert testimony that has the burden of showing that the expert's testimony will be helpful given the facts at hand. *Gopalratnam*, 877 F.3d at 782. Despite Abbott's counsel's forceful arguments, Dr. Makuch's report and deposition testimony do not yet meet this burden.[29]

2. Misapplication of Inclusion Criteria

Assuming that Dr. Makuch can supplement his report and provide sufficient justification for the 75% human-milk-threshold, the court turns to Plaintiffs' other objections to his testimony. Apart from their argument that Dr. Makuch's 75% threshold renders his testimony inapplicable to

[28] As noted, neither of the parties' *Daubert* briefs explain how much CMBF and human milk D.B. (*Etienne* infant) was fed while admitted to the NICU. –Plaintiff's omnibus states that "*none* of the Abbott plaintiffs had *more than* 75% exposure to human milk prior to their onset of NEC," but only provides citations to the record in *Mar* and *Diggs* (where Dr. Makuch's testimony was not offered). (*See* Pls.' Omnibus at 10–11.) They refer to the feeding regimen in "Brown's case," but this refers to K.B., the infant in the *Diggs* bellwether, not D.B., the infant in the *Etienne* case.

[29] Plaintiffs also argue, in the alternative, that Dr. Makuch's 75% threshold resulted in his selecting studies involving cow's-milk-based fortifiers, not cow's-milk-based formula, making his testimony inapplicable in the bellwether cases featuring *only* formula. (Pls.' Omnibus at 11.) This argument is not persuasive. The difference between fortifier and formula, as explained in the court's prior opinion, is a matter of use-case—fortifier adds nutrition to existing milk supply, formula substitutes for milk supply—but the underlying ingredients in cow's-milk fortifier and formula are identical. (*See* supra n.10.) For this reason, Plaintiffs' own expert Dr. Spector made no distinction between fortifier and formula in his analysis, and Plaintiffs' apparent suggestion that Dr. Makuch must do so does not move the court.

the bellwether cases, Plaintiffs further contend that Dr. Makuch did not apply his 75% threshold reliably in selecting studies for his meta-analysis. (Pls.' Omnibus at 12.) Specifically, they charge that Dr. Makuch "cherry-picked" Corpeleijn 2016, a study that found no change in NEC outcomes in higher-CMBF diets, though that study should have been excluded under his 75% threshold criterion. (*Id*. at 12–13.)

To understand Plaintiffs' argument on this score, some background is required. Corpeleijn 2016 was a random control trial testing the difference in NEC outcomes between preterm infants with mother's milk diets supplemented with donor milk and infants with mother's milk diets supplemented with CMBF. (*See* Corpeleijn 2016 [613] at 656.) During the intervention period (the first 10 days of life), the donor milk group was fed 89.1% mother's own milk while the CMBF group was fed 84.5% mother's milk. (*Id*.)[30] After the 10-day intervention period, infants were switched to different diets, presumably according to the physician or parents' discretion and not determined by the study authors. 64.1% of infants in the donor milk group and 56.5% of the CMBF group were switched to an exclusive mother's milk diet after the intervention period. (*Id*.) 11.8% of the donor milk group and 13.6% of the CMBF group were switched to an exclusively CMBF diet. (*Id*.) The remaining infants (24.1% of the donor milk group and 29.9% of the CMBF group) were given an unspecified "mix" of mother's milk, donor milk, and CMBF, likely varying by infant. (*Id*.) The potential outcomes under investigation (sepsis, meningitis, NEC, or death) were measured at 60 days after birth. (*Id*.) The study found a slightly higher incidence of these outcomes (44.7% to 42.1%) in the CMBF group compared to the donor milk group, with 57.7% of all events (58.8% of the CMBF group incidents and 56.8% of donor milk group incidents) occurring within the 10-day intervention period. (*Id*. at 656–57.) There were 17 cases of NEC in both the CMBF group and the human-milk group after 60 days. (*Id*. at 658.) Based on these results, the

[30] For each group, the exact percentage of nutrition provided by donor milk or CMBF was determined by the infant's physician according to nutritional needs and local protocols—the difference in percentage of mother's milk resulted from exigent circumstances, not study design. (*See* Corpeleijn at 656.)

study authors "found no significant effect of pasteurized donor milk during the first 10 days of life for preventing [NEC]" and that the results "stress the importance of providing premature neonates raw milk from their own mother." (*Id*. at 660.)

Plaintiffs contend that Corpeleijn 2016 should have been excluded from Dr. Makuch's analysis under his 75% human-milk threshold because there is no way to determine the percentage of human milk that made up the infants' diets during the entire monitoring period between day 1 and day 60. (Pls. Omnibus at 13.) They describe Corpeleijn 2016 as being divided into two "phases"—"Phase 1" taking place in the first ten days of life, "Phase 2" taking place between days 10 and 60—and note that the study authors do not report the makeup of all infants' diets following the first ten days of the study. (*Id*.) But Plaintiffs' "phase" distinction, which appears nowhere in the study itself, miscasts Corpeleijn's design. The *only* intervention period, during which the assignment of diets to the infant participants was randomized and controlled, was the first ten days of life. As Dr. Makuch explained in his deposition, when asked about Corpeleijn 2016, his "criteria was at the time of the randomization during the period of the primary intervention," meaning that "[Corpeleijn] met [his] criteria." (Makuch Dep. Tr. [612-11] at 369:10–21.) Plaintiffs may object to Dr. Makuch's practice of looking at the diets only during the intervention period, or indeed have objections to Corpeleijn's study design,[31] and may explore these objections in cross examination—but there is no doubt that Corpeleijn reports the percentage of human-milk diets during the intervention period that meet Dr. Makuch's 75% threshold. Dr. Makuch's inclusion of Corpeleijn 2016 does not appear to be a misapplication of his stated criteria.

[31] In the *Mar* litigation, Plaintiffs moved to exclude any reference to Corpeleijn 2016 *in limine* specifically due to its 10-day intervention/60-day monitoring structure, arguing that "the results are not only wholly unreliable, but they are also wrong." (Pl.'s Mot. in Limine [43] in No. 22 C 232.) The court did not rule on this motion prior granting summary judgment.

Plaintiffs' next challenge to Dr. Makuch's method is aimed at his use of the intention-to-treat population in calculating the NEC risk of the respective study groups. (Pls.' Omnibus at 14.) As Plaintiffs note, using the intention-to-treat population (all those randomized) has potential to understate the incidence of NEC in a group—infants that leave the study are counted in the denominator but not in the numerator, should they develop NEC. Moreover, where infants "leave" the formula group of the study or otherwise violate the study protocol, it may mean that they are receiving *more human milk* than the study authors designed—Dr. Makuch's method, therefore, may in fact be counting human-milk fed infants as formula group participants. (*See id*. at 15.) While these are worthwhile topics for cross-examination, Plaintiffs do not appear to contest that using the intention-to-treat population is the gold standard for meta-analytical methods. Moreover, as Abbott points out, Dr. Makuch applied this method consistently and even-handedly—even when doing so was contrary to his ultimate conclusions.[32] Absent allegations that this method is not widely accepted or was applied inconsistently, the court does not find that Dr. Makuch's use of intention-to-treat populations amounted to cherry picking or "fiddl[ing] with the numbers." (*See* Pls.' Omnibus at 14.)

Finally, Plaintiffs argue that Dr. Makuch's cherry-picking was "pervasive." In particular, Plaintiffs identify two studies that they contend met his *a priori* search criteria but were not included in his meta-analysis (Modanlou 1986 and Pettifor 1989) and a third study (Bhat 2003) that was included but self-reported as a case control study (i.e., not an RCT) and did not report the level of NEC observed. (*Id*. at 15.) In the court's view, Plaintffs' concerns here are overstated. Beginning with Modanlou 1986: the study appears to have been properly excluded under Dr. Makuch's criteria because it reports cases of suspected but unconfirmed NEC—not confirmed

[32] Abbott notes that in one of the studies used in Dr. Makuch's meta-analysis, Sullivan 2010, an infant was randomized to the formula group and developed NEC before receiving any formula feedings. (Abbott Resp. [626] at 8; *see* Sullivan 2010 [627-7] at 567.e1.) Under the intention-to-treat method, Dr. Makuch counted this infant as an incidence of NEC in the formula group (that is, suggesting increased risk of NEC from formula) despite the infant having received only human milk prior to the onset of NEC.

NEC (Bell's Stage 2) as required by Dr. Makuch. (*See* Modanlou 1986 [613-3] at 764 ("Two discontinuations in Group II we attributed to clinical (not radiographically proven) suspicion of [NEC].").) Pettifor 1989 appears also to have been properly excluded under Dr. Makuch's 75% threshold—the study reports that infants in the formula group of the study were fed "their own mother's milk mixed in equal volumes with" cow's-milk-based fortifier. (Pettifor 1989 [613-4] at 218.) Finally, regarding Bhat 2003, Plaintiffs are right to note that the authors of the study describe it as a case control study, not an RCT. (*See* Bhat 2003 [613-5] at 30 ("Our case control study . . . .").) They are further correct in noting that Bhat 2003 does not report the level of NEC (suspected or confirmed) observed in the study, referring only to "necrotizing enterocolitis" generally. (*See id*. at 29–30.) But Dr. Makuch has provided adequate reasons for including Bhat 2003 under his criteria despite these ambiguities. First, Dr. Makuch explains that despite the Bhat 2003 authors' own description of the study as a "case control," his review of the study shows that "patients were randomly assigned" to different feeding groups in the study, leading him to conclude that the study was properly understood as an RCT. (*See* Makuch Dep. Tr. at 319:15–21.) Dr. Makuch is not alone in this conclusion; at least one other published literature review (Brown 2020) has also described Bhat 2003 as a "randomised controlled trial." (*See* Brown 2020 [627-18] at 25.) Dr. Makuch further relied on Brown 2020 for his conclusion that Bhat 2003 reported cases of confirmed NEC. (Makuch Dep. Tr. at 298:17–24.)[33] Plaintiffs' perceived inconsistencies in Dr. Makuch's methods, or reliance on Brown 2020, may be bases for cross examination, but they do not reveal methodological infirmities that justify exclusion.

[33] Brown 2020 included Bhat 2003 in its analysis despite requiring that eligible studies report Bell's Stage 2 NEC or higher (Brown 2020 at 6) and Bhat 2003's ambiguity about the level of NEC reported. Brown 2020 also states, however, that it followed up with study authors to address ambiguities or insufficient data. (*Id*. at 7.) It is not unreasonable, therefore, for Dr. Makuch to assume Brown 2020 confirmed that Bhat 2003 reported confirmed NEC when including it in his meta-analysis.

3. Lack of Reproducibility

Plaintiffs argue that Dr. Makuch must be excluded because his method is not reproducible: beyond describing a literature that generated 6,600 articles, Plaintiffs assert, "Dr. Makuch was not able to describe a process or method that any other researcher could duplicate." (Pls.' Omnibus at 16.) Not so. Dr. Makuch's report describes each of his search terms, his predetermined inclusion criteria, and his meta-analysis methods with sufficient detail for another researcher to reproduce his method. *See supra* pp. 25–28. Plaintiffs provide no authority for their suggestion that *Daubert* requires Dr. Makuch to give explanations why each of the 6,600 studies "was included or excluded," or to share the list of results from his initial search. Instead, Plaintiffs cite *Timm v. Goodyear Dunlop Tires N. Am.*, Ltd., 932 F.3d 986 (7th Cir. 2019), where the Seventh Circuit affirmed the district court's exclusion of a motorcycle-accident expert who "conceded that he could point to no empirical data or controlled experiments to support his opinions." 932 F.3d at 994. The expert in that case "had not done any testing to validate his opinion, was unaware of any relevant tests conducted by others, and knew of no way others could objectively replicate his approach." *Id*. *Timm* is a far cry from the facts here; Dr. Makuch has identified the specific studies that support his conclusions and used well-established and replicable meta-analytic methods.

IV. Dr. Brian Smith

Dr. Smith is a neonatologist engaged by Abbott to refute Plaintiffs' general causation testimony and to provide specific causation testimony in the *Diggs* bellwether trial. (*See* Smith Rep. at 1; Abbott Resp. at 11.) Dr. Smith provides both a general opinion that "[b]ovine derived preterm formula and fortifier products do not cause NEC," as well as opinions specific to K.B. (the infant in *Diggs*), including that "[CMBF] did not cause and w[as] not a substantial factor in causing NEC" in K.B., and that K.B. "had multiple conditions associated with NEC, which, together or individually, fully account for his NEC." (Smith Rep. at 2.) In their omnibus motion, Plaintiffs focus their challenge only on Dr. Smith's general causation opinion applicable to all claims against

Abbott. For the purposes of this opinion, therefore, the court only discusses Dr. Smith's general causation opinion without deciding the admissibility of his *Diggs*-specific opinions.

A. Dr. Smith's Report

1. Qualifications

Dr. Smith is the Samuel L. Katz Professor of Pediatrics at Duke University Medical Center. (*Id*. at 1.) He completed his residency in pediatrics in 2004 and fellowship in neonatal medicine in 2007, both at Duke. (*Id*.) He also obtained a Master of Health Sciences Degree from Duke in 2006 and a Master of Public Health degree in biostatistics from the University of North Carolina at Chapel Hill in 2009. (*Id*.) He currently serves as the principal investigator for the Environmental Influences on Child Health Outcomes Coordinating Center at Duke Clinical Research Institute, where he oversees more than 70 clinical sites conducting research on more than 60,000 participants. (*Id*.) He has authored more than 300 peer-reviewed publications on topics ranging from neonatal outcomes, neonatal pharmacology, epidemiology of neonatal infections, and NEC. (*Id*.) In addition to his research, Dr. Smith also maintains a clinical neonatology practice at Duke University Medical Center, where he is responsible for the care of "more than 1,000" infants each year at the hospital's NICU. (*Id*.)

2. Methodology

Dr. Smith performed a systematic literature review to determine whether the epidemiological literature supports a finding of causation between CMBF and NEC. (*Id*. at 5.) As Dr. Smith explains, "[a] systematic review is a well-established method of collating literature that fits pre-specified criteria to answer a specific research question using predefined methods." (*Id*.) His method in performing this systematic review and preparing his report involved four steps: (1) a literature search and application of eligibility criteria, (2) a qualitative assessment and analysis of eligible studies, (3) conclusions based on the analysis of eligible studies, and (4) a review of Plaintiffs' general causation reports. (*See generally id*. at 5–11.)

a. Literature Search

To identify relevant literature for review, Dr. Smith queried the PubMed database of scientific literature using the following search terms: "Breast Milk," "Prolacta," "Donor Breast Milk," "Necrotizing Enterocolitis," OR "Medolac."[34] (*Id*. at 5.) In addition to these terms, Dr. Smith also filtered the results by publication date (1996–2024), language (English only), article type (randomized control trials only), and age of subject (birth–23 months). (*Id*.) He supplemented this PubMed search with a review of the articles cited in eligible articles and of the studies cited by Plaintiffs' experts. (*Id*.) This initial search identified 1,911 studies for further screening. (*Id*.)

Dr. Smith then screened the title and abstract of the 1,911 studies according to predefined eligibility criteria: (1) the patient population of the study was born premature or very low birth weight, (2) one arm of the study (intervention) received primarily human milk or human milk product, (3) another arm of the study (control) received some portion of CMBF or cow's-milk-based fortifier, (4) NEC was tracked as a primary or secondary outcome, and (5) the study was a randomized controlled trial. (*Id*.) After this screening, Dr. Smith identified 20 studies that met his inclusion criteria. (*Id*.) For each eligible study, Dr. Smith created a table recording information including the methods used, the inclusion criteria for participants, how many centers were used to administer the study, the method of blinding, the type of intervention (i.e. what diets were given to infants in different arms of the study), the incidence of NEC observed in each arm, and the Stage of NEC specified in the study. (*Id*. at 5–6; *see also* RCT Analysis, Smith Rep. at A-1–15.) Dr. Smith further reviewed, in addition to the RCTs, any observational studies cited in the eligible RCTs or by Plaintiffs' causation experts. (*Id*. at 6.)

[34] Medolac, similar to Prolacta, is a company that produces human milk-derived infant formula products. Medolac has yet to feature in the parties' briefing regarding the current bellwether cases. *See* Medolac Website, https://www.medolac.com/ (last accessed August 11, 2025.)

b. Analysis of Studies

In analyzing the results, Dr. Smith did not perform a meta-analysis in the manner of Dr. Spector's, Dr. Hedges', or Dr. Makuch's reports. (*Id.*) The reason for this, he explains, is because "studies that are heterogeneous (i.e. different in design and intervention/dose) cannot reliably be combined in a meta-analysis without complicating the interpretation of its results." (*Id.*) Instead, after recording information from each study, Dr. Smith analyzed the result of each individual study according to the following set of qualitative factors.

First, he analyzed each study's efforts to nullify or control for potential confounding factors in reporting their results. (*Id.* at 7.) Dr. Smith notes that there can be a vast range of factors that can impact an infant's chances of developing NEC beyond diet, including gestational age, genetics, distance of mother from the hospital, and concomitant medications. (*Id.* at 7.) RCTs, by their nature, control for many of these factors through randomization, but studies can further control for known confounders through regression analysis. (*Id.*) Crucial to Dr. Smith's conclusions, only four of the 20 eligible RCTs attempted to control for known confounders (Corpeleijn 2016, O'Connor 2018, Nogueira-Pileggi 2022, and Jensen 2024), and of those four, none reported a statistical difference in the incidence of NEC between the study groups. (*Id.*)

Second, Dr. Smith analyzed whether the studies observed a statistically-significant increased incidence of NEC between the different groups—that is, an increase large enough to reject the null hypothesis (he explains that generally, the threshold is a 5% difference). (*Id.*) Dr. Smith accepted the analysis and significance threshold set by the study authors, and observed that out of the 20 RCTs, 17 found no statistically significant association between CMBF and NEC. (*Id.*)

Third, Dr. Smith considered whether each study minimized potential bias by blinding the investigators, caregivers, or parents from group assignments. (*Id.*) Lack of blinding, Dr. Smith explains, can cause various forms of bias, namely attrition bias if parents are not blinded, or detection bias if caregivers or investigators are not blinded. (*Id.*) Evaluating the eligible studies,

Dr. Smith determined that nine were fully blinded, and that only two of these nine (O'Connor 2016 and Colaizy 2024) showed a statistical difference in the incidence of NEC between the groups. (*Id.*)

Fourth, Dr. Smith analyzed which studies chose NEC as the primary outcome; that is, which studies were specifically looking for incidence of NEC in comparing CMBF to human milk diets. (*Id.* at 8.) In doing so, Dr. Smith observed that "most feeding trials examine multiple outcomes" related to growth, neurodevelopment, feeding intolerance, and sepsis in addition to NEC. (*Id.*) In the 20 RCTs he analyzed, almost all identified NEC as a secondary outcome; only one RCT (Adhisivam 2019) used NEC as primary endpoint, and that one found no association between CMBF and NEC. (*Id.*)

Fifth, Dr. Smith scrutinized which studies specified or defined the observed NEC using Bell's staging, which categorizes the severity of NEC based on visual interpretations of abdominal radiographs. (*Id.*) 12 out of 20 eligible RCTs explicitly used Bell's staging language, and of these 12, just three studies (Sullivan 2010, O'Connor 2016, and Colaizy 2024) showed a statistical difference in the incidence of NEC between study groups.

Sixth, Dr. Smith determined whether any of the studies included in his review or referenced in the studies were unpublished, in an effort to determine whether publication bias suppressed studies showing no correlation between CMBF and NEC. (*Id.*) He found no RCTs, but two observational studies, that were not otherwise published but showed no correlation between CMBF and NEC. (*Id.*)

Seventh, Dr. Smith judged whether the studies collected data from a single study center (single-center studies) or from multiple study locations (multicenter studies). (*Id.*) As Dr. Smith explains, while it is possible to administer effective multicenter RCTs involving preterm infant participants, most such studies occur at a single center. (*Id.*) This results in difficulties in interpretation and meta-analysis because of significant differences in both practices and outcomes at different testing centers. (*Id.*) Indeed, among very-low-birth-weight infants treated

in centers within NIH's Neonatal Research Network ("NRN"), incidence of outcome like sepsis and lung disease tends to vary greatly. (*Id*.) Moreover, different centers may use different ventilator types, feeding practices, antibiotic use, and other differences in policy that may, in turn, lead to inconsistent results. (*Id*. at 9.) Multicenter studies, therefore, can account or control for these differences in center-specific policy. Of his eligible RCTs, 11 were multicenter studies; three of them (Sullivan 2010, O'Connor 2016, and Colaizy 2024) demonstrated a statistical association between CMBF and NEC. (*Id*. at 8–9.)

Eighth, Dr. Smith considered the "statistical power" of the studies, or the ability of a study to detect a difference between study groups (where one exists) and avoid false negatives. (*Id*. at 9.) The statistical power of a study depends on two key factors: the sample size of the study and the absolute difference that the study authors are seeking to detect. (*Id*.) Where the null hypothesis of a study requires a large absolute difference to reject, a higher sample size will be required for the study to maintain high statistical power. (*Id*.) A study is considered "powered" for a specific outcome (like NEC) where the study authors ensure that the sample size is sufficient to maintain statistical power given the expected difference in outcomes. Because only one study had been powered for NEC (Adhisivam 2019), and that study found no association between CMBF and NEC, statistical power did not add to Dr. Smith's analysis. (*Id*.)

Finally, Dr. Smith mentions the "randomization" factor—which privileges studies that involve randomizing which infants are placed in the intervention and control groups. (*Id*.) Of course, because he limited his review to RCTs, all the studies he reviewed were randomized.

c. Conclusions

Based on his analysis, Dr. Smith reaches five conclusions about the epidemiological literature testing the relationship between CMBF and NEC. First, he observes that no study compares CMBF to a placebo—thus making it impossible to distinguish whether any observed effect between CMBF and NEC is due to a causal (positive) relation between CMBF and NEC or a causal (negative) relation between human milk and NEC. (*Id*.) Second, he concludes there is

"no consistent trend" across RCTs showing a causal relationship—with 17/20 showing no association. (*Id*.) Third, even accepting Dr. Spector's calculations of the relative risk of CMBF in causing NEC—1.60 or +60%—the relative risk is not strong enough to establish a causal link. (*Id*. at 10.) By comparison, prematurity increases the risk of developing NEC from 0.02% in full-term infants to between 4.6% and 10.4% in preterm infants—a relative risk of 230 to 520. (*Id*.) Fourth, Dr. Smith explains that the authors of studies that *do* observe a statistical association between CMBF and NEC attribute the increased risk to protective attributes of human milk, consistent with Dr. Smith's own publications and the conclusion of the Health and Human Services (HHS) Working Group.[35] (*Id*.) Finally, Dr. Smith remarks that "an increase in comparative risk is not the same as causation." (*Id*.) He notes that existing meta-analyses, even when finding an association between CMBF and NEC, do not conclude that the association is causal. (*Id*. at 11.)

d. Evaluating Plaintiffs' Reports

Dr. Smith concludes his general causation opinions with two criticisms of Plaintiffs' expert testimony. First, he criticizes the use of animal studies to draw conclusions about the mechanism of NEC in humans, an indirect criticism of Dr. Sucre's theories that rely heavily on animal studies. (*Id*.) He opines that "[a]nimal models cannot establish a causal relationship between an exposure and an outcome in humans" because "physiology . . . differs between animals and humans." (*Id*.) Turning to Dr. Spector's report, Dr. Smith criticizes his conclusions for relying on literature that "lacks consistency, is confounded by protection from human milk, demonstrates a very weak association between [CMBF] and NEC . . . and lacks a biologically plausible mechanism." (*Id*.)

[35] In August 2024, the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), at the request of the HHS Secretary, convened an advisory committee of scientists, neonatologists, specialists, and organizational representatives (the "NEC Working Group") to study the relationship between enteral feeding practices and the causes of NEC. (*See* NEC Working Group Rep. [596-1] at i.) Based on the Working Group's findings, the FDA, CDC, and NIH released a consensus statement concluding that "[a]vailable evidence supports the hypothesis that it is the absence of human milk—rather than the exposure to formula—that is associated with an increase in the risk of NEC." (Consensus Statement [596-5] at 2.)

B. Motion to Exclude Dr. Smith

Plaintiffs do not challenge Dr. Smith's qualifications but provide two grounds for the assertion that his report amounts to improper "cherry-picking" of favorable studies. Neither of these criticisms require lengthy discussion. First, Plaintiffs fault Dr. Smith for not selecting search terms related specifically to formula, thus "design[ing] a search strategy that would avoid returning relevant articles about bovine-based formula when conducting his literature review." (Pls.' Omnibus at 17.) But as the court has described, Dr. Smith used broad search terms including "Breast Milk," "Prolacta," "Donor Breast Milk," "Necrotizing Enterocolitis," OR "Medolac" that necessarily would have included any RCTs studying necrotizing enterocolitis—including any studies on the relationship between CMBF and NEC. *See supra* p. 39. Absent evidence of specific, relevant studies that were not discovered by Dr. Smith's search, the mere lack of a specific CMBF-related search term does not amount to "cherry-picking." *See In re Zimmer Nexgen Knee Implant Prods. Liab. Litig.*, No. 11 C 5468, 2015 WL 5050214, at *8 (N.D. Ill. Aug. 25, 2015) (rejecting cherry-picking allegations against expert where "Plaintiff ha[d] not identified a single relevant study that [expert] completely omitted from his review").

Plaintiffs do identify two case control studies that were not included in Dr. Smith's review but that Plaintiffs contend are in fact relevant—Romaine 2018 (co-authored by Dr. Smith) and Johnson 2015. (*See* Pls. Omnibus at 17.) Dr. Smith's literature review, however, was limited to random controlled trials, and only reviewed observational studies to the extent they were cited in Plaintiffs' expert reports or eligible RCTs. *See supra* p. 39. Romaine 2018, despite Plaintiffs' suggestion now, was deemed irrelevant by Plaintiffs' own expert Dr. Spector. (*See* Abbott Ex. 20 [627-20] at 5.) Johnson 2015 is listed as entry 91 in Dr. Smith's list of reviewed studies—he reviewed but did not discuss it. (*See* Smith Rep. at D-7.) Given his explicit focus on RCTs, Plaintiffs may find use in cross-examining Dr. Smith on contrary observational studies, but their exclusion from his report does not suggest that he cherry-picked only favorable studies.

V. Dr. Amanda Starc

Dr. Starc is a health economist engaged by Abbott to opine on the sufficiency of human milk supply to meet the needs of NICUs between 2010 and 2022 (the relevant period of the bellwether claims). (*See* Starc Rep. [613-10] ¶ 8.) Her testimony is expected to be offered in both the *Etienne* and *Diggs* bellwethers.

A. Dr. Starc's Report

1. Qualifications

Dr. Starc is a tenured Associate Professor of Strategy at the Kellogg School of Management at Northwestern University. (*Id*. ¶ 1.) She received her Ph.D. in Business Economics from Harvard University in 2011, and a B.A. in Economics from Case Western Reserve University in 2006. (*Id*.) Prior to joining the Kellogg faculty, she held positions at the Wharton School at the University of Pennsylvania. (*Id*.) In addition to her professorship, she serves as a Research Associate with the National Bureau of Economic Research. (*Id*.)

Dr. Starc's research "examines health care markets"; past research topics include insurance design and pricing, consumer demand for health insurance and healthcare, Medicare reimbursement policies, and the impact of direct-to-consumer advertising of pharmaceuticals. (*Id*. ¶ 2.) She describes her work as "link[ing] demand-side models based on consumer choice and supply-side incentives, using a range of econometric techniques to analyze data." (*Id*.) Her work has been published in leading journals including the American Economic Review, the Review of Economics and Statistics, the Journal of Public Economics, and the Quarterly Journal of Economics. (*Id*. ¶ 2.)

2. Methodology

Dr. Starc's "[a]ssignment" was to determine whether the supply of human milk and human milk-derived products was sufficient between 2010 and 2022 to meet the nutritional demands of preterm infants in NICUs in the United States, such that it would have been feasible for Abbott to replace CMBF with human milk and human milk products. (*Id*. ¶ 8.) Dr. Starc's method in

determining the sufficiency of human milk supply can be divided into three steps: (1) modeling the nutritional demand of preterm infants in NICUs between 2010 and 2022, (2) estimating the supply of human milk in that period, and (3) calculating the shortage of human milk given the modeled supply and demand. (*See generally id*. at 23–52.) Additionally, Dr. Starc's report discusses the feasibility of increasing the supply of human milk in the relevant period to meet a shortfall in supply. (*See generally id*. at 52–68.) Dr. Starc relied on public data from the CDC covering birth statistics and infant deaths, academic research on preterm feeding practices and weight gain up until discharge from the NICU, and industry reports and hospital guidelines detailing preterm feeding protocols. (*Id*. ¶ 43.) In preparing her models and calculations, she was assisted by the staff of Cornerstone Research.[36] (*Id*. ¶ 9.)

a. Modeling Demand

In calculating the volume of human milk needed to meet the needs of preterm infants in U.S. NICUs between 2010 and 2022, Dr. Starc began by categorizing each preterm birth between 2010 and 2022 into a birthweight and gestational age category. (*See id*. ¶ 45.) Dr. Starc assigned each preterm birth between 2010 and 2022 into one of six birthweight categories—ranging from extremely low birthweight (less than or equal to 750 grams at birth) to normal birthweight (greater than 2,500 grams at birth). (*Id*. ¶ 48.) She then subdivided each birthweight category into 20 gestational age categories, ranging from 17 weeks at birth to 36 weeks at birth. (*Id*. ¶ 49.) This resulted in a total of 120 categories of preterm births sorted by birthweight and gestational age, or birthweight-gestational age groups (hereinafter "BW-GA group"). Because the CDC reports the birthweight and gestational age of each birth, Dr. Starc could determine the exact number of preterm births in each BW-GA group between 2010 and 2022. (*See id*. ¶¶ 47–50.)

[36] Cornerstone Research is a research consulting firm that employs experts and assists in the procurement and preparation of expert reports. *See* About Cornerstone Research, https://www.cornerstone.com/about/about-us/ (last accessed June 25, 2025).

For each BW-GA group, Dr. Starc and her team then determined the total nutritional need for a representative infant belonging to that group. First, Dr. Starc calculated a daily nutritional requirement for each representative infant by considering (1) feeding guidelines (specifying required nutrition by birthweight), (2) the rate of weight gain for a representative infant in each BW-GA group, and (3) mortality rate for the representative infant. (*Id*. ¶ 52.) NICU feeding guidelines provide an initial intake rate (in terms of mL of milk per kilogram of infant's weight per day, or mL/kg/day) depending on the infant's birthweight. (*Id*. ¶ 53.) The feeding guidelines also specify a fixed increment at which feeding rate must increase each day (also in mL/kg/day), generally fixing a maximum feeding rate between 150–180 mL/kg/day.[37] (*Id*. ¶ 54.) Using an average from multiple hospital guidelines and academic literature, Dr. Starc determined an average initial feeding rate, incremental feeding increase, and maximum daily feeding rate for each BW-GA group. (*Id*.)

Because the amount of nutrition a preterm infant is provided in the NICU is a function of its weight, Dr. Starc then estimated an average rate of weight gain for preterm infants. Rather than calculate a separate weight growth rate for each BW-GA group, Dr. Starc accepted the "common target" for all premature infants: 15 g/kg/day. (*Id*. ¶ 55.) This made the weight growth rate a constant for each BW-GA group. Combining this constant rate with the daily feeding rates, Dr. Starc was able to calculate a representative infant's daily nutritional requirement (a function of the infant's weight and feeding rate) for each BW-GA group. (*Id*. ¶ 56.) Notably, because both an infant's weight and feeding rate are variable across their time in the NICU, an infant's daily nutritional requirement is also a (multi-variable) function of time. (*See id*.)

Dr. Starc then adjusted her model to account for mortality rates, which necessarily cease nutritional requirements. (*Id*. ¶ 57.) Using CDC's infant mortality data, Dr. Starc calculated the

[37] Though the prescribed feeding rate itself does not increase once this maximum is reached, the nutrition (in mL) continues to increase as the infant gains weight with each day. (*See* Starc Rep. ¶ 54.)

daily rate of mortality (also variable across time) for each BW-GA group and adjusted the daily nutritional requirement for the representative infant of each group accordingly. (*Id.*) Similarly, she adjusted the model to account for infants born before 32 weeks that are not able to receive milk or formula enterally for the first two days after birth (they are given nutrition intravenously)—consistent with literature, Dr. Starc assumed that preterm infants born prior to 32 weeks gestational age receive no milk or formula for the first two days and excluded these days from the model. (*Id* ¶ 59.)

With a daily nutritional requirement for a representative infant in each BW-GA group, adjusted for mortality rates and non-enteral feeding, Dr. Starc then calculated the average length of stay in the NICU for each representative infant—recalling that length of the stay multiplied by daily nutritional requirement determines at the total nutritional requirement for a representative infant. (*Id*. ¶ 60.) The length of an infant's stay in the NICU is a function of the infant's specific health issues and circumstances, but there are strong correlations between an infant's birthweight and gestational age and the length of time that child remains in the NICU. (*Id.*) Dr. Starc thus estimated the average stay in the NICU for each representative infant based on birthweight, gestational age (both determined by their BW-GA group), and birth year. (*Id.*)[38] Dr. Starc specifically used median discharge weights by gestational age as reported by Edwards 2021, which reported data (including discharge weight and gestational age) of preterm infants in NICUs within the Vermont Oxford Hospital Network of member hospitals (a network of hundreds of NICUs worldwide)[39] between 2005 and 2018.[40] (*Id*. ¶ 61.) Using these median values, Dr. Starc

[38] Dr. Starc does not clarify this in her report, but it appears that she further subdivided BW-GA groups into birth years at this stage in the modeling, if she had not done so already.

[39] Vermont Oxford About Us Page, https://public.vtoxford.org/who-we-are-overview/ (last accessed August 13, 2025.)

[40] Applying the values from Edwards 2021 required two extrapolations. First, Edwards 2021 only tracked discharge weights between 2005 and 2018, and Dr. Starc used linear extrapolation to determine median discharge weights by gestational age for 2019–2022 that would

modeled the number of days that a representative infant in each BW-GA group would have stayed in the NICU, for each year between 2010 and 2022. Combining this value with the daily nutritional requirement for each representative infant, Dr. Starc calculated[41] the total nutritional demand of each representative infant, for each year. (*Id.* ¶ 62.)

Finally, Dr. Starc's model arrived at a total nutritional demand for preterm infants between 2010 and 2022 by multiplying the total nutritional demands for each representative infant by the number of infants in each group, for each year. (*Id.* ¶ 64.) This calculation resulted in finding the total nutritional need for preterm infants to range from 1,117,537 mL of milk in 2010 to 1,408,277 mL of milk in 2022, a 26% increase. (*Id.*)[42]

b. Estimating Supply

As Dr. Starc explains, the supply of human milk nationwide is comprised of three components: mother's own milk, donor human milk, and nutritional fortifiers added to human milk to provide necessary nutrition for preterm infants. (*Id.* ¶ 65.) Dr. Starc determined the total supply of human milk between 2010 and 2022 by estimating the supply of these three components during the relevant period and combining them to determine the total.

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be consistent with the 2005–2018 data. (Starc Rep. ¶ 61 n.133.) Second, Edwards 2021 only reported discharge weights of infants born between 24- and 29-weeks gestational age—Dr. Starc thus assumed that infants born at less than 24 weeks had the same median discharge weight of infants born at 24 weeks, and that infants born between 30 and 36 weeks had the same median discharge weight of infants born at 29 weeks. (*Id.* ¶ 61 n.132.)

[41] The court is careful not to oversimplify the nature of this calculation. Because the daily nutritional requirement is a function of time (that is, it varies with each day), it cannot simply be multiplied by the average length of stay in NICU. Rather, Dr. Starc's model would have created a summation formula to calculate and sum the nutritional demand of each day between birth and estimated discharge.

[42] As Dr. Starc explains, this substantial growth in nutritional needs for preterm infants between 2010 and 2022 is driven by an increase in discharge weights and length of NICU stays (presumably both due to improving treatment for preterm infants). (*Id.* ¶¶ 61, 64.)

i. Mother's Milk

Dr. Starc estimated the supply of mother's milk in five steps: (1) within each BW-GA group described in the previous section, she estimated the number of infants fed exclusively mother's milk or some amount of mother's milk (based on a study, described below); (2) for each feeding category, she determined the proportion of the nutritional demand (by %) met by mother's milk; (3) combining the first two steps, for each BW-GA group, she calculated the average contribution of mother's own milk as a proportion of the nutritional demand weighted by the share of infants in each category;[43] (4) she multiplied this average contribution percentage by the total nutritional demand in each BW-GA category; finally, (5) she aggregated the results across BW-GA groups to determine a total estimate of mother's milk supply. (*Id*. ¶ 67.)

At the first step, Dr. Starc relied on Kalluri 2019, a study recording feeding patterns of preterm infants at Boston Medical Center from 2015 to 2017, which found that 34.4% of very-low birth weight ("VLBW") and extremely-low birth weight ("ELBW") infants born before 34 weeks were fed exclusively mother's milk diets—Dr. Starc thus used 34.4% as the proportion of infants in VLBW or ELBW BW-GA groups that were fed exclusively human milk. (*Id*. ¶ 68.) While there was no similar study recording the percentage of moderately-low birth weight ("MLBW") and regular birth weight ("RBW") infants fed exclusively human milk, CDC reports whether an infant was fed any amount of mother's milk prior to discharge from the NICU. (*Id*. ¶ 69.) Dr. Starc was able to use this data to determine what percentage of those ELBW and VLBW infants who received at least some mother's milk in the NICU in fact received exclusively mother's milk. (*Id*.) Applying this same ratio to the MLBW and RBW groups, Dr. Starc estimated that MLBW and

[43] To illustrate this step, if Dr. Starc determined at step (1) that 50% of infants in a BW-GA group—call it "Group A"—were fed exclusively mother's milk and another 50% of infants in Group A were fed some amount of mother's milk, and determined at step (2) that "exclusively" meant that 100% of nutritional demand was met with mother's milk and "some" meant that 50% of nutritional demand was met with mother's milk, Dr. Starc would determine at step (3) that mother's milk met 75% (50% x 100% + 50% x 50%) of the nutritional demand of Group A.

RLBW groups were fed 36.8% and 36.9% exclusive mother's milk diets, respectively. (*Id.*) To determine what percentage of infants in each group were fed some, but not exclusively, mother's milk, Dr. Starc subtracted the number of infants estimated to be fed exclusively mother's milk from the total number of infants in each group that received any mother's milk, per CDC. (*Id.* ¶ 70.)

At step two, Dr. Starc defined exclusive mother's milk diets as meeting 100% of the nutritional demands of the infant with mother's milk, and the "some" mother's milk diet as meeting 50% of the nutritional demand with mother's milk.[44] (*Id.* ¶¶ 68, 70.) At step three, Dr. Starc multiplied the percentage of infants in each feeding category by the percentage of nutritional demand met by mother's milk in each feeding category—this resulted in, for each BW-GA group, a percentage of the total nutritional demand that was met by mother's milk. (*Id.* ¶ 71.) Multiplying these values by the total nutritional demand (in mL) for each group, at step four, Dr. Starc arrived at the total volume mother's milk supplied to meet nutritional demand for each BW-GA group. (*Id.*) Summed across all groups, Dr. Starc estimated the total mother's milk supply between 2010 and 2022, which ranged from 550 million mL in 2010 to 760 million mL in 2022. (*Id.*)

ii. Donor Milk

Donor milk can be sourced from both non-profit milk banks and for-profit companies; Dr. Starc determined the supply of each between 2010 and 2022. (*Id.* ¶ 72.) In American NICUs, Dr. Starc noted, most donor milk is provided by non-profit milk banks accredited, and subject to the reporting of, the Human Milk Banking Association of North America ("HMBANA"). (*Id.* ¶ 73.) HMBANA is a non-profit that serves as a source for providing guidelines for safety and FDA compliance for non-profit milk banks. (*Id.*) Among its various functions, HMBANA distributes

[44] As explained in her deposition, Dr. Starc arrives at this 50% assumption by relying on studies observing feeding habits among mothers in the NICU, which tend to alternate between mother's milk and formula feedings. (*See* Starc Dep. Tr. at 229:3–7.) She acknowledges that "there's not an article [that has] clear evidence of . . . the exact precise number." (*Id.* at 231:4–9.) Given that percentages of mother's milk in an infant's diet is likely to have a wide distribution, Dr. Starc concluded that "50 percent [was] the most reasonable choice" for an estimate. (*Id.* at 231:10–18.)

press releases reporting the total amount of donor milk distributed by its member milk banks. (*Id*. ¶ 74.) Because HMBANA includes milk banks in Canada (that distribute donor milk only to Canadian hospitals), Dr. Starc estimated and subtracted the amount of donor milk distributed by Canadian milk banks from the total reported by HMBANA.[45] (*Id*.) HMBANA's 2018 press release states that just 77% of its donor milk distributions were allocated to NICUs; relying on that information, Dr. Starc further subtracted the proportion of donor milk that was distributed to individuals rather than NICUs. (*Id*.) As a result, Dr. Starc estimated that the supply of donor milk from HMBANA milk banks rose steadily from under 50 million mL in 2010 to 200 million mL of donor milk in 2022. (*Id*. ¶ 76.)

To account for other sources of donor milk (that is, for-profit companies producing human-milk derived formulas or fortifiers and hospitals using in-house milk banks), Dr. Starc relied on a 2020 survey of U.S. medical directors that reported that 80.9% of NICUs relied on nonprofit milk banks, 17.1% sourced donor milk from Prolacta and Medolac (for-profit companies producing donor milk-derived products), and 2% relied on in-house milk banks and Ni-Q (a different human milk product company) products. (*Id*. ¶ 77.) Dr. Starc thus assumed that all premature infants in the 19.1% of NICUs reported in the survey met their demand from for-profit and in-house donor milk sources and added this volume to the supply of donor milk. (*Id*.)[46] Finally, she applied a 2%

[45] Dr. Starc estimated the amount of donor milk distributed in Canada by taking the number of HMBANA member banks in Canada as a proportion of all members and subtracted this percentage from the total donor milk reported by HMBANA. (Starc Rep. ¶ 74.)

[46] Dr. Starc's report does not clearly explain how exactly this survey of NICUs provided her with a value for commercial donor milk supply, or otherwise how this 19.1% value was incorporated into her donor milk supply estimate. At her deposition, she stated that she used the information from the 2020 survey to "inflate the amount of donor milk available from HMBANA banks," but does not explain if this was done by adding an absolute value of commercial donor milk or multiplying the non-profit supply by an estimated adjustment. (*See* Starc Dep. Tr. at 259:5–24.)

spoilage rate[47] to the entire estimation of donor milk, across non-profit and commercial sources. (*Id*. ¶ 78.)

iii. Fortifier Enhanced Nutrition

The addition of fortifiers to human milk not only adds crucial nutrients for preterm development, but also adds volume to the human milk supply that is available; thus, Dr. Starc estimates the increase in volume provided by fortifiers as a form of human milk supply. (*Id*. ¶ 79.) Using the mixing guidelines provided by human-milk-based and cow's-milk-based fortifier products, Dr. Starc estimated that fortifiers increase the volume of mother's milk and donor milk by 22.6%. (*Id*.) To account for the fact that not all hospitals use fortifier products for human milk, Dr. Starc multiplied the additional supply created by introducing fortifier by 82.2%—the percentage of NICUs reported to incorporate fortifiers by a 2018 study. (*Id*. ¶ 80 n.159.)

iv. Total Human Milk Supply

Combining her estimates of available mother's milk and donor milk, and incorporating the increase in volume due to the addition of nutritional fortifiers, Dr. Starc calculated the total supply of human milk in U.S. NICUs. She determined that the supply increased from 707 million mL in 2010 to 1.2 billion mL in 2022. (*Id*. ¶ 82.) Most of this supply came from mother's milk, which made up 77.8% of the human milk supply in 2010, declining to 63.7% by 2022. (*Id*.) Donor milk, in contrast, increased as a proportion of the total supply from 6.5% in 2010 to 20.7% in 2022. (*Id*.)

c. Calculating Shortage

Having modeled both the demand for preterm infant nutrition and the supply of human milk in the period between 2010 and 2022, Dr. Starc then calculated the "gap" between demand and supply. From Dr. Starc's estimations, the gap was widest in 2010, where the total demand

[47] Dr. Starc determined this spoilage rate from the abstract of a 2018 presentation to the Association of Women's Health, Obstetric and Neonatal Nurses, which reported a decrease in donor-milk-spoilage rate from 43% to 1.5% with an improved storage process used at one NICU. (Starc Rep. ¶ 78.) Dr. Starc based her "conservative" 2% spoilage rate on this presentation. (Starc Dep. Tr. at 264:7–265:2.)

for preterm nutrition (1.117 billion mL) exceeded the total supply of human milk (707 million mL) by over 400 million mL of milk. (*Id*. ¶ 85.) While Dr. Starc's model shows that this gap decreased each year, by 2022, the gap between supply and demand still exceeded 215 million mL of milk. (*Id*.) To put this gap in terms of infants left without access to human milk, Dr. Starc's model shows that 61,191 preterm infants lacked such access to human milk in 2010 and 32,065 preterm infants lacked such access in 2022. (*Id*.) Based on these calculations, Dr. Starc opines that "the supply of human milk and human milk products was insufficient to meet the nutritional demand of premature infants in U.S. NICUs from 2010 through 2022." (*Id*. ¶ 87.)

d. Economic Feasibility

After determining that a gap existed between preterm nutritional demand and human milk supply, Dr. Starc's report analyzes the feasibility of increasing the human milk supply between 2010 and 2022. The analysis begins by noting that the limited supply of mother's milk results from several non-economic factors, including lactation difficulties, separation from the infant, and challenges in producing sufficient breast milk. (*Id*. ¶ 89.) Dr. Starc goes on to observe that NICUs already engage in many initiatives to educate mothers on the benefits of supplying breast milk, and she sees no evidence that the supply of mother's milk could be increased. (*Id*.) As for donor milk, Dr. Starc posits that expanding the supply of donor milk could be achieved by either "increasing the incentives for milk donation or reducing the barriers and costs to donation." (*Id*. ¶ 90.) Dr. Starc concludes, however, that attempts to financially incentivize mothers to contribute more donor milk are unlikely to meet the demand of U.S. NICUs, for three reasons: (1) circumstances of the donor milk market make the supply of donor milk relatively inelastic, (2) a sharp increase in supply of donor milk would dramatically increase costs to hospitals, and (3) the U.S. lacks regulation that would ensure that additional donor milk supply would be accessible to at-risk VLBW and ELBW infants. (*See generally id*. ¶¶ 91–113.)

In a lengthy section of her report, Dr. Starc identifies several characteristics of the donor milk market that make supply particularly resistant to financial incentives and market demand.

First, the literature shows that women tend to donate to non-profit milk banks for altruistic, as opposed to financial reasons, meaning that the supply is less responsive to financial incentives. (*Id*. ¶ 92.) Second, the supply of donor milk is constrained by the fact that only lactating women with surplus milk can donate milk, and only a percentage of these women can produce *enough* surplus to meet the minimum donation thresholds required by many milk banks. (*Id* ¶¶ 95–96.) Third, there are financial and non-financial barriers to milk donation, including required screenings, geographical barriers due to the location of milk banks, and opportunity costs to women of pumping, storing, and shipping of milk for donation. (*Id*. ¶¶ 97–99.) These factors, taken together, lead Dr. Starc to conclude that the supply of donor milk is unlikely to increase in response to incentives or market demands. (*Id*. ¶ 100.)

Dr. Starc observed, further, that an increase in donor milk supply between 2010 and 2022 would have imposed significant costs on hospitals. She notes that while preterm infants are the final consumers of donor milk, hospitals are primary payors for donor milk—according to one study, 86% of surveyed hospitals reported that donor milk is paid for from the hospital's budget. (*Id*. ¶ 102.) Donor milk costs four to fourteen times more than CMBF. (*Id*.) Assuming the same price for donor milk at a higher supply,[48] meeting the full demand for infant nutrition with donor milk could be cost-prohibitive for hospitals. (*Id*. ¶¶ 104–05.)

Finally, Dr. Starc notes a lack of nationwide infrastructure or regulation that would ensure efficient allocation of donor milk to preterm infants even if the supply of donor milk were to increase. In the absence of nationwide regulation, individual hospitals tend to have different protocols as to how donor milk is allocated for its infant population; some prioritize donor milk recipients by birthweight, others by gestational age. (*Id*. ¶ 108.) It is thus difficult to determine

[48] Dr. Starc does not explain why she assumes that the cost for donor milk would remain the same should the supply dramatically increase. While she notes that demand for donor milk would increase if CMBF were taken off the market, driving the price higher (Starc Rep. ¶ 104), she does not consider whether the price would be driven down if there were a significant increase in supply.

with any confidence whether an increase in donor milk supply would, in fact, result in VLBW or ELBW infants receiving donor milk. Moreover, the lack of nationwide infrastructure on distributing donor milk means that the supply of donor milk is distributed unevenly across geography—hospitals that are located near milk banks or in states with milk bank programs are more likely to have access to milk than hospitals located far from a source of donor milk, regardless of absolute supply. (*Id*. ¶ 110.)

Taking these factors together, Dr. Starc concludes that, to a reasonable degree of scientific certainty, "it would not have been feasible to use human milk and human milk products instead of cow's milk-based preterm formula to meet the nutritional demand of premature infants in U.S. NICUs from 2010 through 2022." (*Id*. ¶ 116.)[49]

B. Motion to Exclude Dr. Starc

Plaintiffs seek Dr. Starc's exclusion on two grounds. First, they argue that Dr. Starc has insufficient facts and data to make several key assumptions in her analysis. (*See* Pls.' Omnibus at 19–20.) Second, and more fundamentally, they argue that Dr. Starc's report is really a product of researchers employed at Cornerstone Research, "with her input being little more than her signature at the end of the report." (*Id*. at 19, 21–22.)

1. Insufficient Facts and Data

Rule 702 requires that an expert's "testimony is based on sufficient facts or data." FED. R. EVID. 702(b). An expert's testimony satisfies this requirement when "the expert considered sufficient data to employ the methodology" and there is "a rational connection between the data and the opinion." *Manpower*, 732 F.3d 796, 808–09 (7th Cir. 2013) (quoting *Stollings v. Ryobi Techs., Inc*., 725 F.3d 753, 766 (7th Cir. 2013)). Rule 702(b) does not require a thorough

[49] The court observes that Dr. Starc's report does not discuss the possibility of donor milk as a feasible replacement for *some* of the nutritional demand of preterm infants met by CMBF, or the possibility of increasing the supply of donor milk in specific markets.

appraisal of the quality of the underlying facts; "an expert's reliance on faulty information is a matter to be explored on cross-examination; it does not go to admissibility." *Id*.

Plaintiffs do not take issue with Dr. Starc's general reliance on CDC-published data, academic articles, and hospital guidelines in reaching her conclusions. *See supra* pp. 45–46. Rather, they base their 702(b) argument on four purportedly "incorrect" assumptions Dr. Starc made in formulating her economic models, asserting that Dr. Starc lacked sufficient data to justify those assumptions. (*See* Pls.' Omnibus at 19–20.) First, they argue that Dr. Starc's extrapolations of Edwards 2021 (described *supra* p. 51) to calculate the average length of stay of infants in the NICU are not based on sufficient data because "Dr. Starc did not consult with any neonatologist, doctor, or anyone else to verify that the extrapolated assumptions she used were correct." (Pls.' Omnibus at 19.) Second, they argue that Dr. Starc's estimation for the percentage of infants that are fed exclusively mother's milk (*see supra* p. 51) improperly relies on studies of women historically less likely to feed their children mother's own milk,[50] faulting her for "never r[unning] any sensitivity analyses to ensure that her calculations . . . were reliable." (*Id*. at 20.) Third, Plaintiffs argue that Dr. Starc's assumption that infants fed "some" amounts of mother's milk were given a 50% diet (*see supra* p. 51) was not reliable because "Dr. Starc did not consult with any neonatologist or pediatric nutritionist to verify that her assumption was correct." (*Id*.) Finally, Plaintiffs assert that Dr. Starc made an "incorrect assumption" of the spoilage rate of donor milk based on a "poster presentation" (*see supra* p. 52), but do not appear to argue that the presentation is faulty or unreliable. (*Id*.)

[50] Specifically, Plaintiffs observe that Kalluri 2019, the study Dr. Starc relies on for assuming that 34.4% of VLBW and ELBW infants are fed exclusively mother's own milk, had a study population of 47% non-Hispanic Black mothers—a population that Dr. Starc recognizes tends to breastfeed at a lower rate and is not representative of the U.S. at large. (*See* Starc Dep. Tr. at 205:8–208:9.) Kalluri 2019's population would be representative if non-Hispanic Black mothers accounted for 47% of all preterm births, but that does not appear to be the case. *See* March of Dimes Preterm Birth Stats, https://www.marchofdimes.org/peristats/data?reg=99&top=3&stop= 63&slev=1&obj=1 (last accessed August 11, 2025.)

Plaintiffs have raised valid objections to assumptions that make up some, but not all, of the inputs of Dr. Starc's models. The court notes, however, that these objections are properly targeted at the *correctness* of Dr. Starc's assumptions, not the sufficiency of the factual basis. For example, while Plaintiffs assert that Dr. Starc's estimation of the percentage of infants fed exclusively mother's milk is potentially skewed by biases in the studies she relies on, they do not contest Dr. Starc's testimony that these studies are "best in class data that is available." (Starc Dep. Tr. at 215:8–216:4.) Indeed, nowhere do Plaintiffs suggest that Dr. Starc relied on sources outside the scope of her field, or that there were more accurate sources for these assumptions that she ignored. As the court has laid out in its description of Dr. Starc's report, Dr. Starc makes a rational connection between a reputable source and each one of the many inputs for her model. Plaintiffs may disagree with her reasoning in arriving at certain inputs or argue that she should have double-checked her assumptions with another expert, but the fact that "the reasoning behind [a] choice [of inputs] could be challenged as incomplete or faulty does not make it any less grounded in real data." *Manpower*, 732 F.3d at 809. Testing the accuracy of key assumptions is the province of cross examination, not 702(b). *See Stollings*, 725 F.3d at 768 ("The fact that an expert's testimony contains some vulnerable assumptions does not make the testimony irrelevant or inadmissible.")

2. Over-Reliance on Cornerstone Employees

Plaintiffs argue that Dr. Starc must be excluded because she is serving as a mere "mouth-piece" for the non-testifying employees of Cornerstone Research who conducted much of the coding underlying her report. (Pls.' Omnibus at 20.) They note that Dr. Starc admitted to outsourcing the coding behind her models to Cornerstone employees "Nick" and "Eduardo," who checked each other's work in preparing the code, but Dr. Starc could not testify to their level of education or training. (*See* Starc Dep. Tr. at 91:3–93:3.) Importantly, Plaintiffs seek exclusion on this ground under Federal Rule of Civil Procedure 26(a)(2)(B), which requires that expert

disclosures in discovery "be accompanied by a written report—prepared and signed by the witness." FED. R. CIV. P. 26(a)(2)(B).

There is little caselaw supporting Plaintiffs' theory that Rule 26(a)(2)(B) can be used as a sword to exclude experts that rely, perhaps over-rely, on the work of their assistants; every case cited by Plaintiffs involves a circumstance where an expert's report is excluded because it was prepared by *counsel*. *See, e.g., Bekaert Corp. v. City of Dyersburg*, 256 F.R.D. 573, 578 (W.D. Tenn. 2009) ("Whether an expert report was prepared in a manner consistent with the mandates of Rule 26, usually turns on whether *counsel's participation* so exceeds the bounds of legitimate assistance[.]") (emphasis added); *Manning v. Crockett*, No. 95 C 3117, 1999 WL 342715, at *3 (N.D. Ill. May 18, 1999) ("[Counsel] preparing the expert's opinion from whole cloth and then asking the expert to sign it if he or she wishes to adopt it conflicts with Rule 26(a)(2)(B)[.]"); *Trigon Ins. Co. v. United States*, 204 F.R.D. 277, 292 (E.D. Va. 2001). There is no allegation here that Abbott's counsel ghost-wrote or otherwise contributed to Dr. Starc's report—indeed, there is no allegation that anyone other than Dr. Starc drafted the report, and Plaintiffs cite no case in which an expert's testimony was excluded because of reliance on work performed by assistants or co-workers.

Plaintiff's concern has little weight here, as Dr. Starc has testified that the work performed by Cornerstone employees—including data collection, data cleaning, and help with literature reviews (Starc Dep. Tr. at 78:14–23)—was done entirely "under [her] direction." (*See id*. at 93:10–17; 142:18–143:10.) Indeed, Dr. Starc has clarified that the type of support she received from the Cornerstone employees in preparing her report was no different than the support she receives from assistants in her academic research. (*See id*. at 29:19–23; 79:12–23; 83:20–22.) As another court has noted, "[Rule 26(a)(2)(B) does not suggest that an expert cannot appropriately rely upon others to help, and it would be unrealistic to conclude otherwise." *Brand v. Comcast Corp., Inc*., 215 (N.D. Ill. 2014). And there is "nothing remarkable about a paid expert preparing a report with the assistance of staff." *Manpower*, 732 F.3d at 810. Plaintiffs' suggestion that "Dr. Starc cannot

explain how her reliance materials were compiled because she did not do the work" is directly contradicted by her deposition in which Dr. Starc explained precisely how data was collected and compiled for use in her models. (Starc Dep. Tr. at 164:8–168:13.) Plaintiffs had an opportunity, in Dr. Starc's deposition, to demonstrate that she lacked familiarity with the facts, methods, or conclusions of her report—they failed to do so.

CONCLUSION

Plaintiffs' omnibus motion to exclude expert testimony [612, 613] is granted in part and denied in part. Portions of Dr. Hedges' report based on his miscalculation of Sullivan 2010 are excluded pending a supplemental report correcting the error. Dr. Makuch's report is excluded subject to a supplemental justification for his 75% human milk criteria. Plaintiff's objections to Defendants' expert opinions are otherwise overruled.

ENTER:

Date: August 15, 2025

REBECCA R. PALLMEYER
United States District Judge